A DISSERTATION

ON

ANALYTICAL STUDY OF MRI BRAIN IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA PATIENTS”

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R. MEDICAL UNIVERISTY

CHENNAI

with partial fulfillment of the regulations

for the Award of the degree

M.S. [OBSTETRICS & GYNECOLOGY]

Branch – II

DEPARTMENT OF OBSTETRICS & GYNECOLOGY

STANLEY MEDICAL COLLEGE

CHENNAI

APRIL - 2017

CERTIFICATE

This is to certify that the dissertation entitled **"ANALYTICAL STUDY OF MRI BRAIN IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA PATIENTS**" submitted by **Dr. KIRITHA RANJANI. A.C**, to the faculty of Obstetrics and Gynaecology, Stanley Medical College, The Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirements for the award of M.S Degree (**OBSTETRICS & GYNECOLOGY**) , is a bonafide research work carried out by her under our direct supervision and guidance.

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DECLARATION

I, **Dr. KIRITHA RANJANI.A.C** solemnly declare that dissertation titled, **"ANALYTICAL STUDY OF MRI BRAIN IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA PATIENTS"** is a bonafide work done by me at Govt. RSRM Lying in Hospital, Stanley Medical College ,Chennai ,under the guidance and supervision of Professor and Head Of the Department Dr.K.Kalaivani MD.,DGO., The dissertation is submitted to Tamilnadu Dr. M.G.R. Medical University, towards partial fulfillment of university rules and regulations for the award of M.S. Degree (Branch – II) in Obstetrics & Gynecology.

Place : Chennai.

Date :

Dr. KIRITHA RANJANI.A.C,

INSTITUTIONAL ETHICAL COMMITTEE,
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Title of the Work : Analytical study of MRI Brain in Antepartum and postapartum Eclampsia patients.

Principal Investigator : Dr. A C Kiritha Ranjani

Designation : PG, MS (O & G)

Department : Department of O & G
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 24.03.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

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STANLEY MEDICAL COLLEGE
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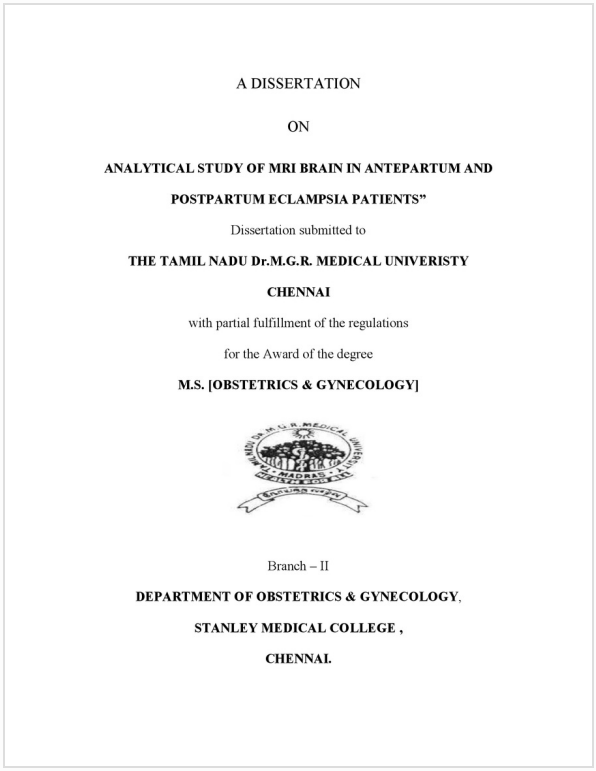

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INTRODUCTION

Hypertensive disorders are one of the most common complications of pregnancy.

It is one of the major cause of maternal and perinatal mortality and morbidity worldwide .Among the Hypertension disorders, Preeclampsia and Eclampsia are a life threatening multisystem disorder, which affects Cardiovascular, Hematological, Renal, Hepatic, and Central nervous system.

Cerebrovascular involvement is the direct cause of death in 40% of the Gestational Hypertension patients. When it is diagnosed and intervened at the earlier, symptoms and radiological changes can be reversed.

Besides clinical presentation, Neuroimaging is the only route to find out the CNS involvement. It provides a more accurate assessment of degree of CNS involvement.

Vascular pathology in the organs, is the main stay of subject of this study. Understanding the effect of Pregnancy and Postpartum state altering the structure and function of cerebrovascular system gives an important clue about emergence of Eclampsia.

Neurovascular changes and complication occurring as a result of vascular pathology in cerebrovascular system is the reason behind Eclampsia. When it is identified by imaging studies and intervened, we can prevent the complication becoming irreversible.

To identify the prevalence of neurovascular complications and neurovascular changes in Eclampsia, a prospective and retrospective analytical study was conducted in Government RSRM Lying in

hospital,Stanley Medical College, Chennai. MRI Brain was done for 50 patients of Eclampsia and the findings were analysed.

AIM OF THE STUDY

Analytical study of MRI BRAIN in Antepartum and Postpartum Eclampsia patients.

To do MRI BRAIN for all Antepartum and Postpartum Eclampsia patients and to identify the cause ,to arrive at a proper diagnosis and further management.

To intervene as soon as the cause is made to avoid maternal morbidity and mortality.

To identify the prevalence of neurovascular complications in these cases.

REVIEW OF LITERATURE

Hypertension is one of the dreaded complication, resulting in Maternal, Fetal morbidity and mortality. It is a multiorgan failure mainly affecting cerebrovascular system endangering the life of the patient.

Pregnancy is related with cardiovascular adaptation according to the needs of mother and baby,both in systemic and local circulation.The changes in cerebral blood flow autoregulation and cerebrovascular resistance increases the permeability of BLOOD BRAIN BARRIER(BBB), resulting in hydrostatic edema.

DEFINITION

Eclampsia, which is considered as a complication of severe Preeclampsia ,is defined as ,New onset of grand mal seizure activity And/or unexplained coma during pregnancy or postpartum in a woman with signs or symptoms of preeclampsia.

It typically occurs during or after the 20^{th} week of gestation or in the postpartum period.

Preeclampsia is defined as Hypertension associated with Proteinuria, where hypertension is defined as a blood pressure of 140mmHg systolic or more and diastolic of 90mmHg or more, measured 6 hrs apart with proteinuria of more than 300mg/24hrs or >1+ on dipstick (30mg/dl or

more) after 20 weeks of pregnancy in a previously normotensive and non proteinuric woman.

CLASSIFICATION

NATIONAL HIGH BLOOD PRESSURE EDUCATION PROGRAM WORKING GROUP (2000)

GESTATIONAL HYPERTENSION

1) BP >/= 140/90 mmHg for first time during pregnancy.
2) No proteinuria.
3) BP returns to normal < 12 weeks postpartum.
4) Final diagnosis made postpartum.
5) May have other signs and symptoms of pre-eclampsia
   for example, epigastric discomfort or thrombocytopenia.

PRE-ECLAMPSIA

Minimum criteria:

BP >/= 140/90mm of Hg after 20 weeks gestation.

Proteinuria>/=300mg/ 24 hours or >/= 1+ dipstick

Increased certainity of Pre-eclampsia:

BP >/= 160/100mm of Hg

Proteinuria 2gm/24 hours or 2+ dipstick.

Serum creatinine > 1.2mg/dl unless known to be previously elevated.

Platelets < 1,00,000/mm3

Microangiopathic hemolysis (increased LDH)

Elevated ALT or AST

Persistent headache or other cerebral or visual disturbances.

Persistent epigastric pain.

ECLAMPSIA

Seizures that cannot be attributed to other causes in a woman

with pre-eclampsia.

SUPER IMPOSED PRE-ECLAMPSIA (ON CHRONIC HYPERTENSION)

New onset proteinuria >/=300mg/24 hrs in a known hypertensive

women without proteinuria occuring before 20 weeks of gestation.

Sudden increase in proteinuria or blood pressure or

platelet count <1,00,000/mm3 in women with

hypertension and proteinuria before 20 weeks gestation.

CHRONIC HYPERTENSION

BP > 140/90 mm Hg before pregnancy or diagnosed

before 20 wks gestation not attributable to gestational

trophoblastic disease.

Or

Hypertension first diagnosed after 20 weeks gestation

and persistent after 12 weeks postpartum.

PROTEINURIA

Proteinuria is an important sign of preeclampsia.It helps in assessing the severity of preeclampsia.The mechanism behind proteinuria is vasospasm of afferent glomerular arterioles leading to anoxic damage to endothelium of glomerular tuft resulting in endotheliosis. It will result in increased capillary permeability causing leakage of proteins, tubular

reabsorption is equally depressed. Significant proteinuria is described as 300mg/l or more of urinary protein loss in 24 hours or persistent 30mg/dl (1+ dipstick) in random clean catch sample on atleast 2 occasions collected 6 hours apart. Dipstick method is routinely used to measure proteinuria and colour changes indicates

PROTEIN

Trace - 0.1gm/l

1+ - 0.3gm/l

2+ - 1.0gm/l

3+ - 3.0gm/l

4+ - 10gm/l

INCIDENCE

The incidence of preeclampsia is 7-10% depending on the population studied. The incidence of eclampsia in Govt RSRM Lying in Hospital, Chennai is 0.5% Worldwide approximately 50,000 women are estimated to die annually because of eclampsia.The overall maternal death rate of eclampsia is 2% .

The perinatal mortality among babies born to eclamptic mother was 32% compared to 10.5% for Total perinatal mortality.

PATHOPHYSIOLOGY

The underlying basic pathology in Eclampsia & Pre eclampsia is endothelial dysfunction and intense vasospasm affecting almost all vessels particularly Uterus, Kidney, Placental bed and Brain.

There is increase in circulatory pressor substances and increased sensitivity of vascular system to normally circulating pressor substances.

Normally there is an invasion of endovascular trophoblasts into walls of spiral arterioles of uteroplacental bed. In first trimester, endovascular trophoblasts invades upto decidual segments.In second trimester it invades upto myometrial segments. This process replaces endothelial lining and muscular wall by fibrinoid formation, finally spiral arterioles becomes distended, tortuous and funnel shaped. Thus spiral arterioles changes into low resistance low pressure high flow system.

In Preeclampsia, there is a failure of second wave of endovascular trophoblast migration and there is reduction of blood supply to fetoplacental unit.

In Preeclampsia there is imbalance in different components of prostaglandins, relative or absolute deficiency of vasodilator,PGI2 from vascular endothelium, and increased synthesis of thromboxane, a potent vasoconstrictor of platelets.

Increased vascular sensitivity to Angiotensin II and also Angiotensinase activity is depressed due to proteinuria with elimination of alpha 2 globulins.

Nitric oxide is synthesized from L-arginine from vascular endothelium and syncytiotrophoblast, which helps in vasodilation and anti platelet aggregation. It prevents intervillous thrombosis. Deficiency of Nitric oxide contributes to hypertension.

Endothelin-1 is synthesized by endothelial cells and is a potent vasoconstrictor contributing to the cause of hypertension.

Cytokines, Interleukins are derived from activated leukocytes leading to endothelial injury.

Abnormal lipid metabolism results in oxidative stress. Lipid peroxides, ROS, and superoxide radicals cause endothelial injury and dysfunction.

Factor V leiden mutation is one of the cause.

Immunological response of mother to increased circulating levels of VCAM-1 resulting in the formation of immune complexes in placenta, maternal serum and other organs.

Extravillous trophoblasts expressing reduced amounts of nonclassic HLA G are prone to become preeclamptic.

In case of T helper cells ,Th1 action is increased and Th1/Th2 ratio changes which leads to enhanced immunologically mediated inflammatory reaction, triggered by placental microparticles and adipocytes.

Polymorphisms of genes for Fas receptor, hypoxia inducible factor -1 alpha protein (HIF-1α) Protein,IL-1β,Lymphotoxin-α,TGF-β3, apoE, are associated with preeclampsia.

Circulating endothelial cells and circulating microparticles (CEC,CMP) are elevated fourfold in preeclamptic women.

Increased serum levels of soluble endoglins and Soluble Fms-like tyrosinase kinase 1(sFlt-1) which are antiangiogenic disrupt endothelial function and release inflammatory mediators resulting in preeclampsia.

John and colleagues (2002) showed in general population, a diet high in fruits and vegetables that is having antioxidant activity decreases blood pressure.

Coagulation abnormalities is due to increased activation & consumption of platelets & low antithrombin III levels, abnormal prostaglandins metabolism & in few cases florid DIC.

These changes lead to hypertension, edema,proteinuria, bleeding tendency, renal dysfunction, liver damage & neurological abnormalities.

CEREBROVASCULAR PATHOPHYSIOLOGY:

The Brain is protected from extremes of blood pressure by an autoregulation system that ensures constant perfusion, over a range of systemic pressure, in response to systemic hypotension, cerebral arterioles dilates to maintain perfusion. Above the limits of auto regulation, hypertensive encephalopathy may occur.

The classic microvascular lesion is fibrinoid necrosis of the arterial wall, perivascular microinfarcts, and hemorrhages. Major lesions are subcortical edema, multiple non hemmorhagic areas of softening in the entire brain and hemmorhagic areas in white matter.

The hemorrhages are also seen in pons and basal ganglia which may rupture into ventricles.

First theory, Trommer (1988) proposed that in response to acute and severe hypertension, cerebrovascular overregulation leads to vasospasm. Resulting hypoxia leads to ischaemia and CYTOTOXIC EDEMA.

Second theory, when the systemic blood pressure rises above the cerebral autoregulatory pressure forced dilation and vasoconstriction of vessels takes place. At capillary level, it disrupts end-capillary pressure causing increased hydrostatic pressure, hyperperfusion,

and extravasation of plasma and red cells through endothelial tight junction openings, leading to VASOGENIC EDEMA.

Hinchey (1996) ,with imaging studies,these changes present as Posterior reversible leukoencephalopathy syndrome.

Cipolla(2014),proposes that PRES is most common in parietooccipital cortex,boundary zone of anterior, middle and posterior cerebral arteries. In most situations,these lesions aree reversible.

Recent hypothesis suggests that syndrome results from breakthrough of autoregulation with passive overdistension of cerebral arterioles.It would result in interstitial extravasation of protein and fluid causing focal vasogenic (hydrostatic edema) in peripheral vascular distribution of involved vessel.

In eclampsia, presumably due to loss of autoregulation of cerebral blood flow, there was hyperperfusion similar to hypertensive encephalopathy, women with headache have increased cerebral perfusion.

Apollon and co-workers 2000, Cunningham and Twickler 2000 Concluded that Eclampsia is due to transient loss of cerebral autoregulation resulting in widespread hypodense areas confirmed by MRI.

Zeeman et al found in normal pregnancy cerebral blood flow decreases early in pregnancy and is 20 percent less than non pregnant value in late pregnancy. Their investigations showed that preeclampsia ,there is hyperperfusion leading to vasogenic edema which is identified using MRI.

The regional heterogeneity of sympathetic vascular innervations explains the susceptibility of posterior circulation in pathology.

Experimental studies proposed that sympathetic innervation of the intracranial arterioles is shown to protect the brain from marked blood pressure. Ultrastructural studies shows that Internal carotid system is much better supplied with sympathetic innervations than in Vertebrobasilar system.

Posterior circulation is mostly affected because perivascular sympathetic nerves protect anterior circulation but not the posterior.

Preeclampsia is termed as "TOXEMIA OF PREGNANCY" presents with various histologic features. During autopsy, these findings are usually made out.

There are two types of pathology seen in brain as a result of eclampsia.

GROSS HEMMORHAGE:

Severe hypertension causing rupture of arteries resulting in gross hemorrhage.

FOCAL LESIONS:

Sometimes it presents with more widespread, focal and seldom fatal lesion. They are edema, hyperemia, ischaemia, thrombosis and hemorrhage.

As cerebral hypoperfusion mostly affects parietal and occipital lobes, headache and stomata usually presents.

Sibai(2005) and Zwart(2008), 50-75% of women present with headaches,20-30% presents with visual changes preceding eclamptic convulsions. intensity of headache varies from mild ,severe, intermittent to constant, which usually does not respond to analgesia but responds to Magnesium sulfate regime.

Meldrum (2002), convulsions are caused by excessive release of glutamate which causes massive depolarization of network neurons, and bursts of action potentials causing extended seizures resulting in brain injury .

(Cunningham,1995) states that blindness is rare with preeclampsia alone, but it complicates eclampsia in 15%of women.

Blindness manifests about a week or more after delivery (chamber,2004)

Final and worse presentation is GENERALISED CEREBRAL EDEMA, which present with mental status changes, varying from confusion to coma. The most dreaded complication of this is Tentorial Herniation.

Various imaging modalities are available to diagnose the lesion.

- MRI of Brain
- CT
- MR ANGIOGRAPHY
- TRANSCRANIAL DOPPLER STUDY
- SPECT

MRI OF BRAIN:

Magnetic Resonance Imaging (MRI) is well known for its far more superior soft tissue contrast and multiplanar resolution when it is compared to CT scan. It is good at diagnosing hemorrhage, ischaemia and edema.

In case of cerebaral edema usually CT reports normal study. When in case of MRI it demonstrates transient T2 lesions in suncortical regions of parieto occipital lobes.

Brainstem and/or basal ganglia which are rarely involved is also diagnosed with MRI.

Raps 1993 and Bartynski 2003, proposed that, In case of late postpartum eclampsia, CT usually shows low density reas. But in MRI it shows positive T2 MRI .

Morriss et al found that minimal changes are found on T2 MRI in 2/10 of severely preeclamptic women based on increased signal intensity compared to abnormal findings in T2 signal intensity in women with eclampsia. these patients present with symptoms of headache, vertigo, or visual disturbances.

At times persistent hyperintense T2 lesions actually represent permanent brain lesions consistent with infarctions.

Diffuse Weighted Magnetic Resonance Imaging and Apparent Diffusion Coefficient mapping (DWI/ADC)

Two different types of cerebral edema, vasogenic and cytotoxic are differentiated by using neuroimaging techniques. Vasogenic edema is due

to increased hydrostatic pressure and capillary leak. Cytotoxic edema usually presents with cell death.

It is impossible to differentiate between these forms between CT and MRI techniques. With series of MR acquisitions, Diffusion Weighted Imaging Sequences and Apparent

Diffusion coefficient maping, it is possible nowadays to characterize T2 MRI images with hyperintensity in eclamptic women using (DWI/ADC)

DWI technique works on the principle of Quantification of diffusion of free water, which is found to be decreased in ischaemic brain tissue .Thus ischaemic brain regions can be identified within minutes to hours after onset of neurological symptoms. This appears as area of high signal intensity compared with signal from normal brain. After an ischaemic event evaluation, there is a shift of water from extracellular to Intracellular space which results in restricted diffusion and therefore reduced ADC values.

It mainly results from decreased Na+ K+ ATP ase activity in the glial cell membranes and therefore results in decrease in water molecule transporting causing cell death. These areas represents cytotoxic edema and or visualized as hyperintense lesions on DWI. Thus regional ADC values is an important noninvasive tool for monitoring the

evaluation of time and to identify spatial expansion of the ischaemic lesions.

Reversible vasogenic edema can be identified by a combination of normal DWI with high T2 signal lesions and increased ADC.

Studies using additional diffusion weighted imaging sequences found the origin of brain edema is mainly vasogenic but can be ischaemic or cytotoxic in an eclamptic patients. In such cases, DWI hyperintense lesions with decreased ADC is present in a case of vasogenic edema.

Characteristic findings in MRI of Brain are hyperintense lesions on T2 weighted, Fluid Attenuation Inversion Recovery (FLAIR) Or proton weighted sequences which usually affects the grey and white matter of occipital or parietal lobes.

Other areas affected are deep white matter structures, basal ganglia and white matter of frontal or temporal lobes and brain stem.

Louiero 2003, Zeeman 2004 explained advantage of taking MRI in eclampsia patients. It appears that 20-25 % of women with eclampsia explains the lesions are consistent to be cerebral infarction (Sheehan 1973, Richards 1988). It has to be mentioned that all these

women were normotensive as well as asymptomatic at time of the follow up.

Longstreth 1996, Bernick found that, Subclinical infarcts and white matter lesions in general are associated with an increased risk of clinical stroke events, physical limitations and cognitive impairment such as dementia.

Trommer 1988, Lewis 1988, Will 1987 proposed that vasospasm presents in MRA as diffuse or multifocal segmental narrowings or vasospasm of the cerebral vasculature.

Kanayama 1993, Ito 1995, instead of intracranial hemorrhage, vasospasm may be the only finding in eclampsia which may be persistent upto two weeks postpartum.

Recent experiments found out that vasospasm appears as sausage string pattern in angiography.

In few eclamptic women, sudden death occurs concurrently with convulsions or shortly after convulsions.It happens mostly in case of massive cerebral hemorrhage.

Older women with chronic hypertension presents with cerebral hemorrhage due to long standing hypertension induced lipohyalinosis of small and medium sized arteries. In that case

striatocapsular area, thalamus, cerebellum and brain stem are the sites most frequently affected, Imaizumi 2004.

Alternatively, Salerni et al 1988 describes that cerebral infarction may transform into a hemorrhagic infarction in case of young nulliparae presenting with HELLP syndrome.

Another rare cause of intracerebral hemorrhage is rupture of aneurysm or arteriovenous malformation.

In subrachnoid hemorrhage a small amount of blood is seen over the convexity of the frontal/parietal lobes extending into the sylvian fissure or interhemispheric tissue.

With Conventional angiography ,ruptured arterio venous malformation or intracranial aneurysm, or cortical venous sinus thrombosis can be made out.

Shah 2003, Gregory 2003, proposed that Subarachnoid hemorrhage in eclampsia is the result of rupture of cortical petechiae over the surface of the brain or rupture of small pial veins. This type of subarachnoid hemorrhage seems to carry a benign prognosis.

Giannina describes a subdural hematoma associated with thrombocytopenia in an eclamptic women ,which spontaneously developed. Uncal herniation is an another cause of death of the patient.

Biller 1995 found out that in an eclamptic patient with hematoma in right basal ganglia which transformed into an abscess due to secondary infection of episiotomy through bacterial dissemination.

PRES

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES) also known as **REVERSIBLE POSTERIOR LEUCOENCEPHALOPATHY SYNDROME (RPLS)** is hypothesized to be the primary injury based on its clinical pathological as well as neuroimaging features. In non pregnant patients PRES may occur after a subacute elevation of blood pressure. Areas of cerebrovascular vasodilation and vasoconstriction may both coexist in the acute phase of hypertension. The clinical presentation is variable and may include headache, seizures, visual changes, altered mental status and occasionally focal neurological signs.

PATHOLOGY:

The endothelial cell dysfunction is thought to play a dominant role in the pathogenesis of PRES. Endothelial injury leading to altered

regulation of cerebrovascular response for the release of systemic vasoactive metabolites like AT II as well as to the release of Endothelial vasoactive substances resulting in hyper activity of blood vessels and labile blood pressure.

MRI findings include hyperintensities on T2 weighted images. Three patterns described on MRI imaging include, superior frontal sulcus pattern, Dominant parieto-occipital pattern and Holo hemispheric watershed pattern.

Treatment is based on the cause. Control of blood pressure may alleviate the symptoms. Many cases will resolve within one to two weeks of controlling the blood pressure and eliminating the triggering factors. Yet uncommon death may occur due to intracerebral hemorrhage or cerebral edema. PRES may occur in 5-10% of cases.

Optic Changes in Eclamptic Patients

Visual disturbances are seen in 40% of eclampsia patients. They include scotomota, amaurosis, blurred vision, diplopia, chromatopsia or homonymous hemianopsia (Weiner 1987). The pathology behind the visual disturbance / blindness is retinal edema and vascular changes such as retinal arteriolar vasospasm or thrombosis of the central retinal artery or retinal detachment.

Moseman 2002 stated that with newer MRI imaging modality ,lateral geniculate body edema along with focal cerebral edema can be made out.These features lead to cortical blindness.

Cortical blindness

Cortical blindness presents with intact pupillary light reflexes ,intact ocular movements and normal ophthalmologic findings which excludes the peripheral cause of blindness.

Transient blindness is calculated to occur in about 1-15 % (Cunningam 1995, Torres 1995) in eclampsia patients. This is seen in MRI as T2 hyperintense lesions .

Lesions are seen particularly in the parieto-occipital area which are supplied by the posterior circulation, and are reversible in follow up imaging studies.

It is usually confirmed by diffusion MRI which shows the absence of restricted diffusion in the presene of vasogenic edema.

So far, only three women have been described in the literature demonstrating permanent blindness. Two of them had a combination of both abnormal retinal findings consistent with Purtschers retinopathy and MR evidence of brain infarcts located in the lateral geniculate nuclei Blodi 1990, Moseman 2002.The third patient with persistent blindness

did not undergo MR imaging and therefore no conclusions can be drawn, Lara 2002. Two additional case reports deserve mention. Delefosse describes a case of transient cortical blindness in a woman who was 26 days postpartum. The authors concluded that retained placental fragments may be associated Delefosse 2003.

Finsterer describes a preeclamptic patient who suffered transient cortical blindness after nitroglycerin administration. MR imaging with diffusion sequences demonstrated a typical picture of vasogenic edema. The authors conclude that nitroglycerin may aggravate the development of vasogenic edema secondary to enhancement of cerebrovascular vasodilation and may result in cortical blindness

MATERIALS AND METHODS

TYPE OF STUDY

Prospective and Retrospective Analytical study**.**

PERIOD OF STUDY

September 2014 to August 2016

SETTING

The study was conducted at Govt. Stanley medical college. The study was approved by the board of ethical committee.

SAMPLE SIZE

50

METHODOLOGY

SCREENING

MRI Brain is done for all antepartum and postpartum eclampsia patients after stabilising them.

PROSPECTIVE STUDY

After getting consent ,MRI Brain is done for all antepartum and postpartum eclampsia patients and the findings are analysed.

Duration: Jan 2016 - August 2016.

RETROSPECTIVE STUDY

Case sheets and MRI Brain reports of all antepartum and postpartum eclampsia patients are taken from MRD after obtaining permission from Superintendent and MRD incharge.

Duration: Sep 2014-Dec 2015.

INCLUSION CRITERIA

Antepartum eclampsia

Postpartum eclampsia

EXCLUSION CRITERIA

Epilepsy

Cerebral tumours

Preexisting renal disorders.

CONSENT

Informed consent in the form of written consent was obtained from the patients or relatives (in situations where patients is indisposed) after explaining the procedure.

DATA ANALYSIS AND RESULTS

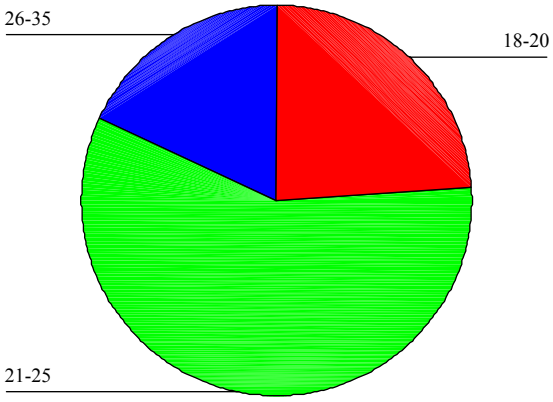
MRI Brain was taken for 50 patients with Eclampsia .MRI findings and data collected from the 50 patients are analysed.

AGE IN YEARS

TABLE-1: AGE DISTRIBUTION

Age in years	Frequency	Percent
18-20	12	24.0
21-25	29	58.0
26-35	9	18.0
TOTAL	50	100.0

Age in years

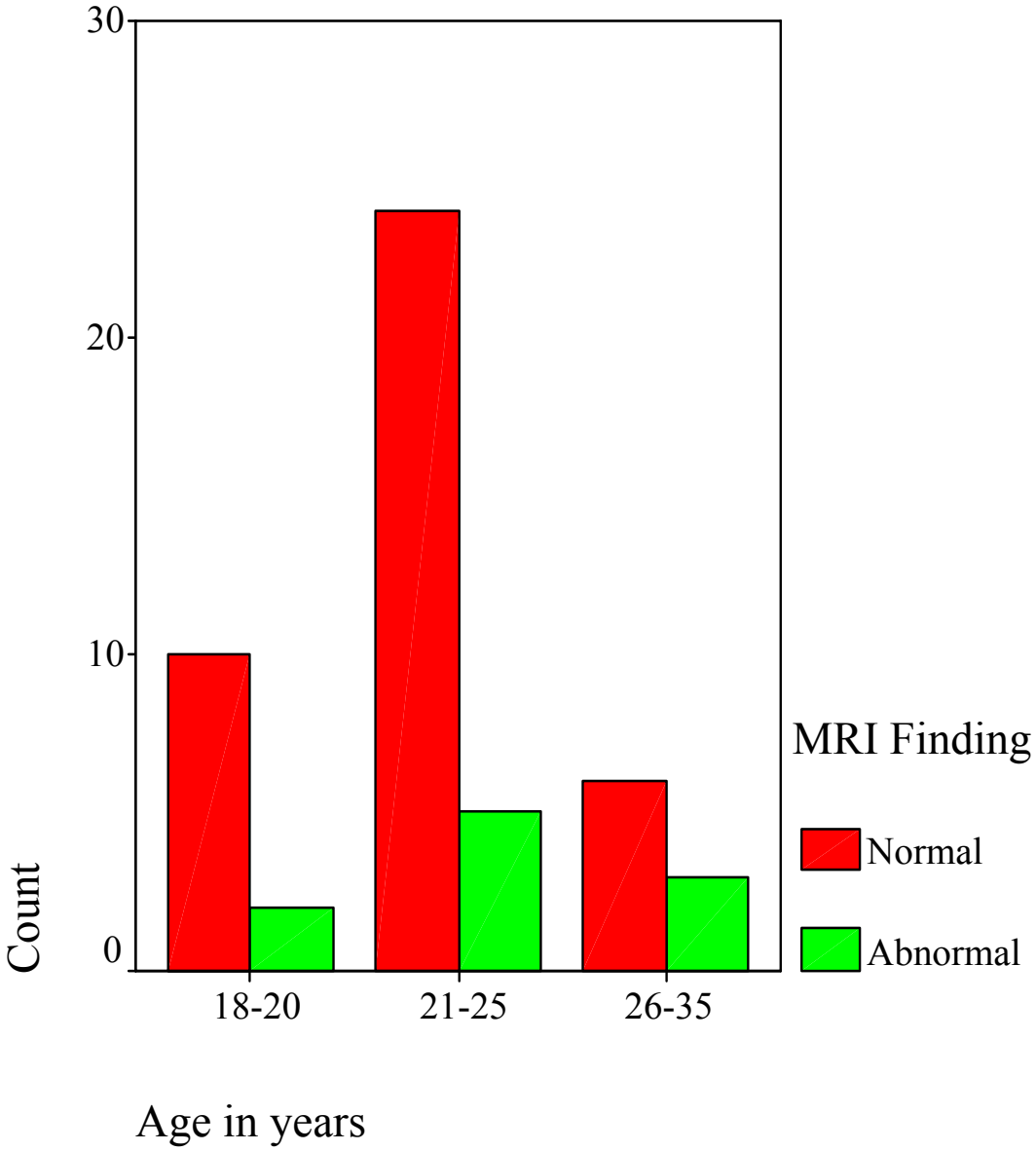


The most common age group in our study is in the range of 21-25 yrs. 29% of the patients were in the range of 21-25 yrs. Only 9% of the patients were in the age group of more than 25 years.

AGE IN YEARS * MRI Finding

Age in years		MRI Finding		Total	P value
		Normal	Abnormal		
18-20	Count	10	2	12	
	% within Age in years	83.3%	16.7%	100.0%	
	% within MRI Finding	25.0%	20.0%	24.0%	
21-25	Count	24	5	29	
	% within Age in years	82.8%	17.2%	100.0%	
	% within MRI Finding	60.0%	50.0%	58.0%	0.543
26-35	Count	6	3	9	
	% within Age in years	66.7%	33.3%	100.0%	
	% within MRI Finding	15.0%	30.0%	18.0%	

50% of the patients having abnormal MRI finding falls under the age group of 21-25 yrs, which is statistically not significant (p value is 0.543).

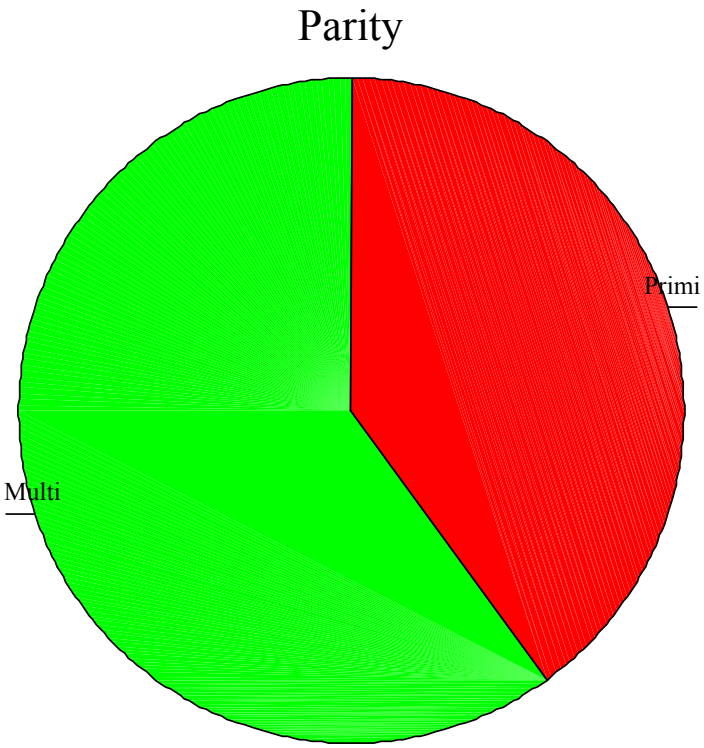

30
20
10
0
Count
18-20
21-25
26-35
Age in years
MRI Finding
Normal
Abnormal

PARITY

TABLE-2: PARITY

Parity	Frequency	Percent
Primi	20	40.0
Multi	30	60.0
Total	50	100.0

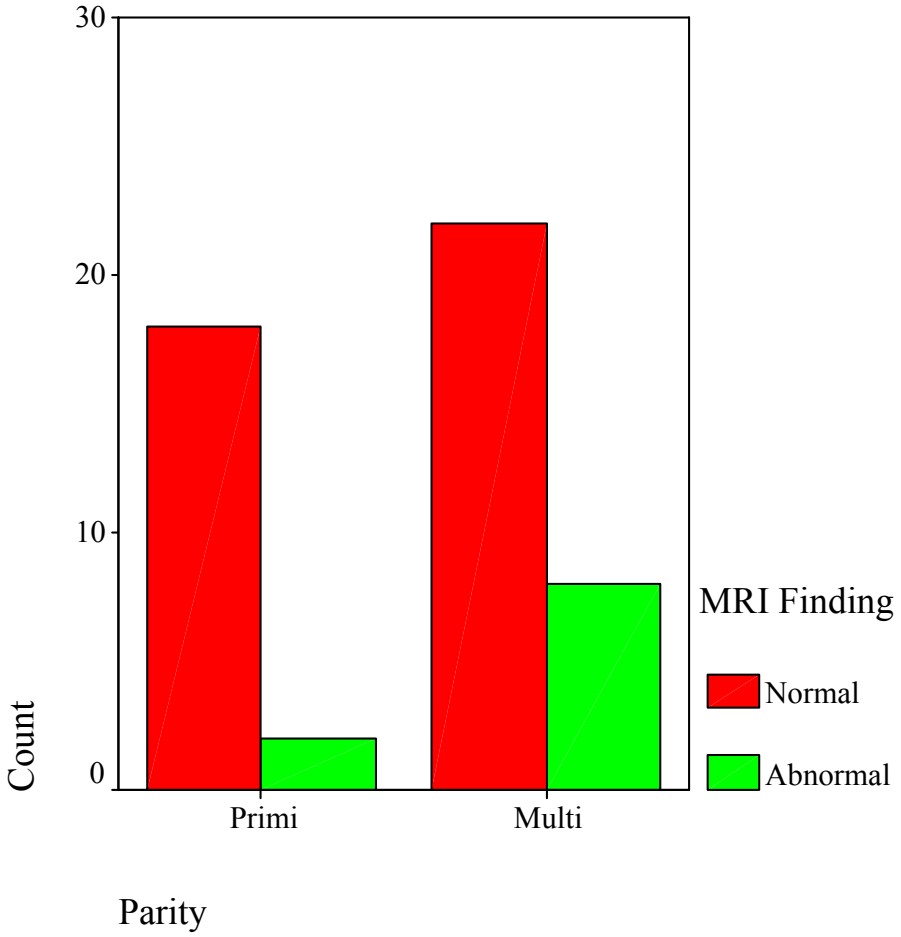
Multigravida constitutes the majority in our study.60% of the patients were multigravida.



PARITY* MRI FINDING

Parity		MRI Finding		P value
		Normal	Abnormal	
Primi	Count	18	2	0.139
	% within Parity	90.0%	10.0%	
	% within MRI Finding	45.0%	20.0%	
Multi	Count	22	8	
	% within Parity	73.3%	26.7%	
	% within MRI Finding	55.0%	80.0%	

In my study group , 80% of the patients who have abnormal MRI findings were Multipara which is not statistically significant (p value 0.139)


30
20
10
0
Count
Primi
Multi
Parity
MRI Finding
Normal
Abnormal

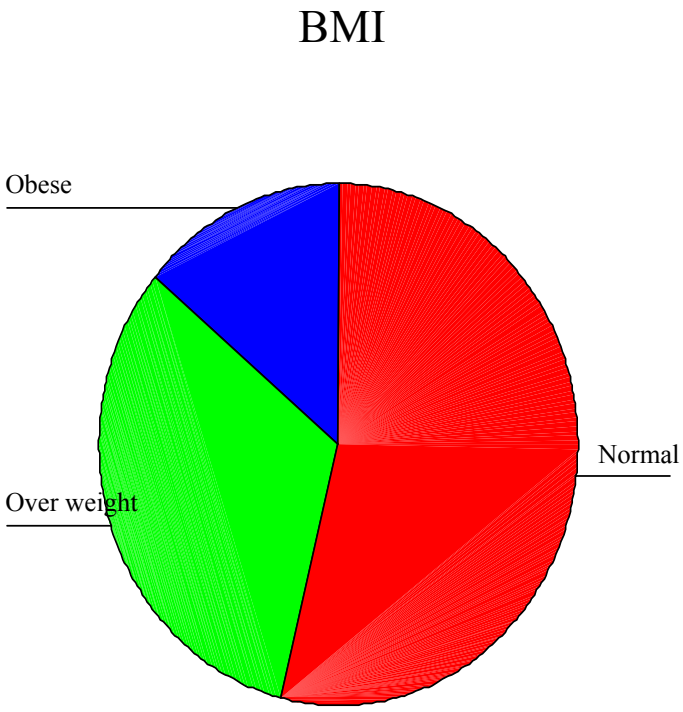
.

BODY MASS INDEX

TABLE-3: BODY MASS INDEX

BODY MASS INDEX	Frequency	Percent
Normal	27	54.0
Over weight	16	32.0
Obese	7	14.0
Total	50	100.0

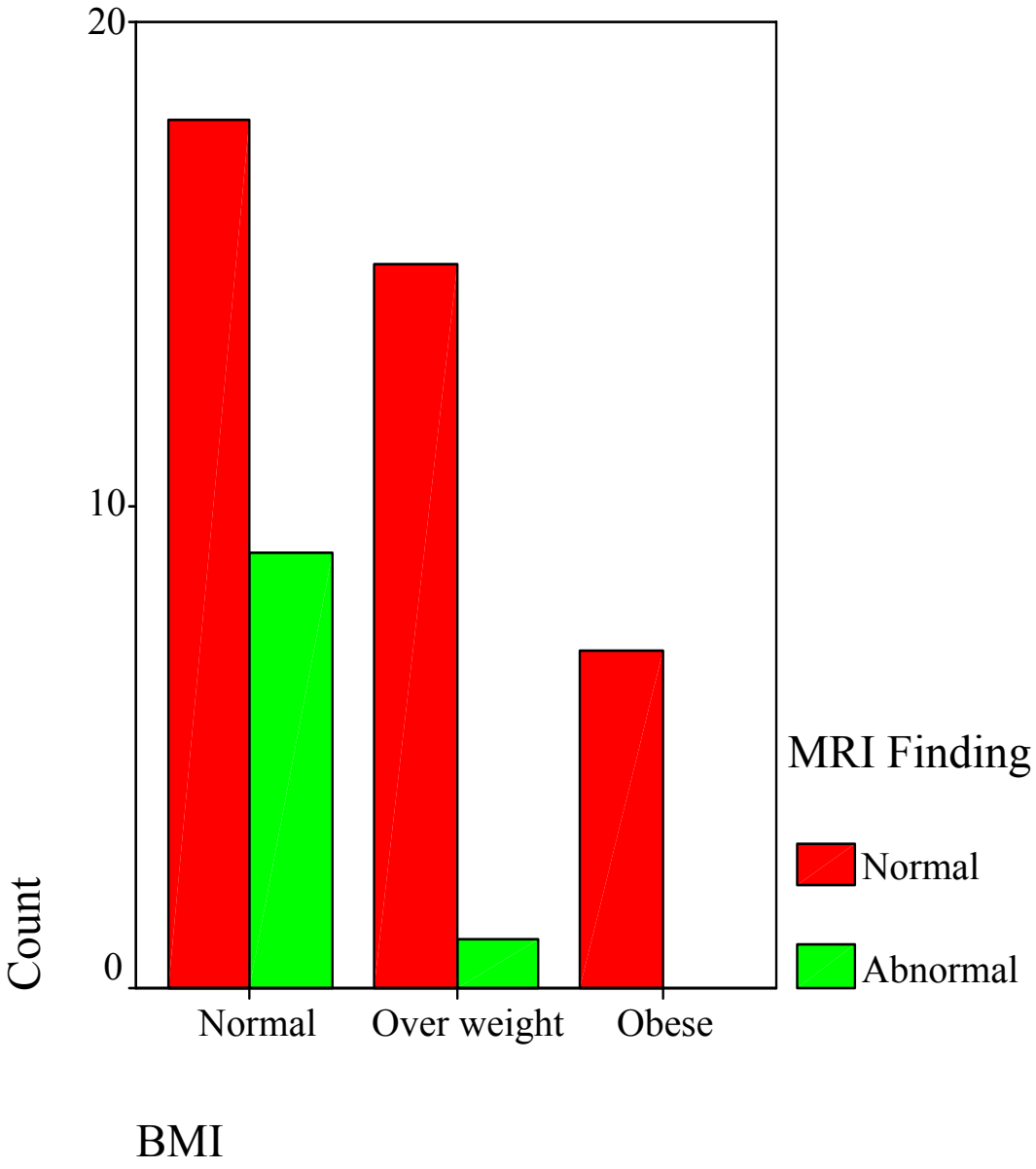
In my study group,54% of the patients have normal BMI.14 % of the patients are obese.



BMI * MRI Finding

			MRI Finding		
			Normal	Abnormal	P value
	Normal	Count	18	9	.036*
		% within BMI	66.7%	33.3%	
		% within MRI Finding	45.0%	90.0%	
	Over weight	Count	15	1	
		% within BMI	93.8%	6.3%	
		% within MRI Finding	37.5%	10.0%	
	Obese	Count	7	0	
		% within BMI	100.0%	.0%	
		% within MRI Finding	17.5%	.0%	

90% of the normal BMI patients have abnormal MRI findings.
p value 0.036* which is statistically significant.


20
10
0
Count
Normal
Over weight
Obese
BMI
MRI Finding
Normal
Abnormal

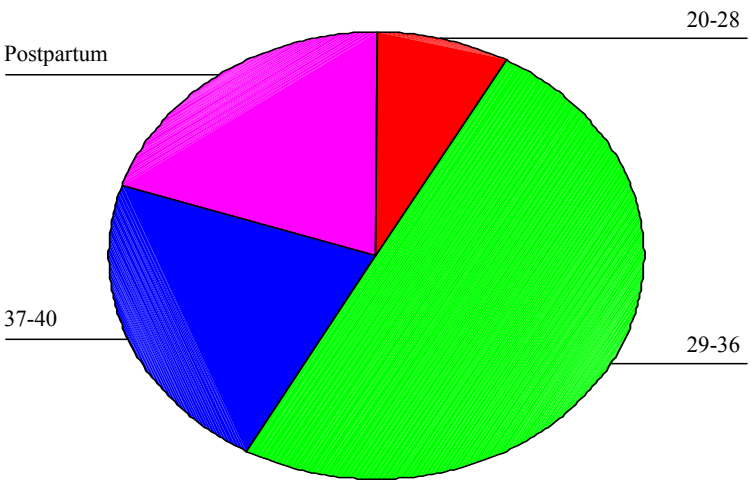
GESTATIONAL AGE (in weeks)

TABLE-4: GESTATIONAL AGE

GA in weeks	Frequency	Percent
20-28	4	8.0
29-36	25	50.0
37-40	11	22.0
Postpartum	10	20.0
Total	50	100.0

Most of the patients were in the antenatal period in my study group at the time of presentation In my study group 50% percent of the patients falls between 29-36 weeks of gestational age.10% are postpartum patients.

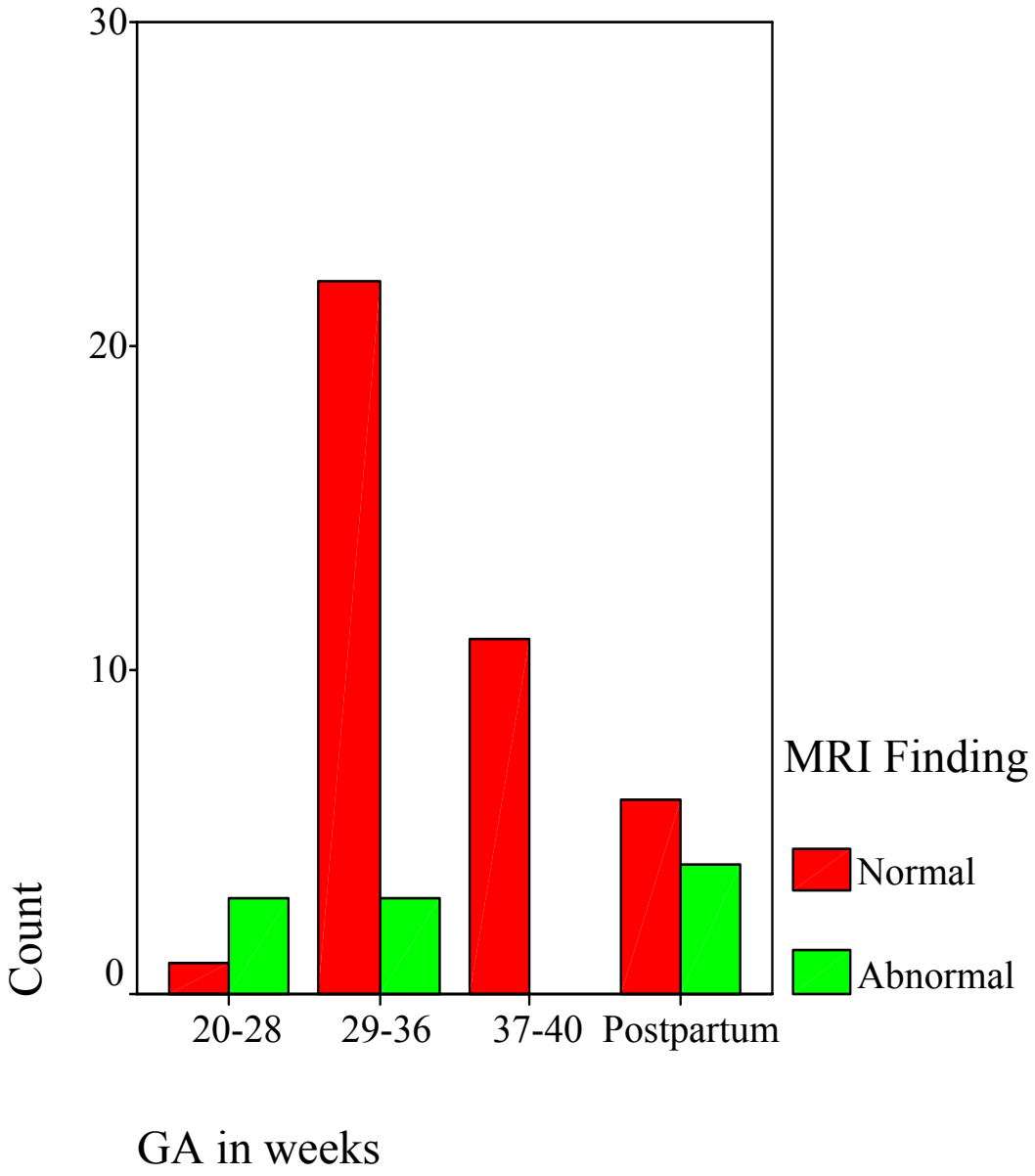
GA in weeks



GESTATIONAL AGE IN WEEKS * MRI FINDING

			MRI Finding		P value
			Normal	Abnormal	
GA in weeks	20-28 Weeks	Count	1	3	.003**
		% within GA in weeks	25.0%	75.0%	
		% within MRI Finding	2.5%	30.0%	
	29-36 Weeks	Count	22	3	
		% within GA in weeks	88.0%	12.0%	
		% within MRI Finding	55.0%	30.0%	
	37-40 Weeks	Count	11	0	
		% within GA in weeks	100.0%	.0%	
		% within MRI Finding	27.5%	.0%	
	Postpartum	Count	6	4	
		% within GA in weeks	60.0%	40.0%	
		% within MRI Finding	15.0%	40.0%	

In our study group, the gestational age between 20-28 weeks and 29-36 weeks have 30% of abnormal MRI findings each. But in the postpartum group, 40% of the patients have abnormal MRI findings. p value is 0.003, Significant.

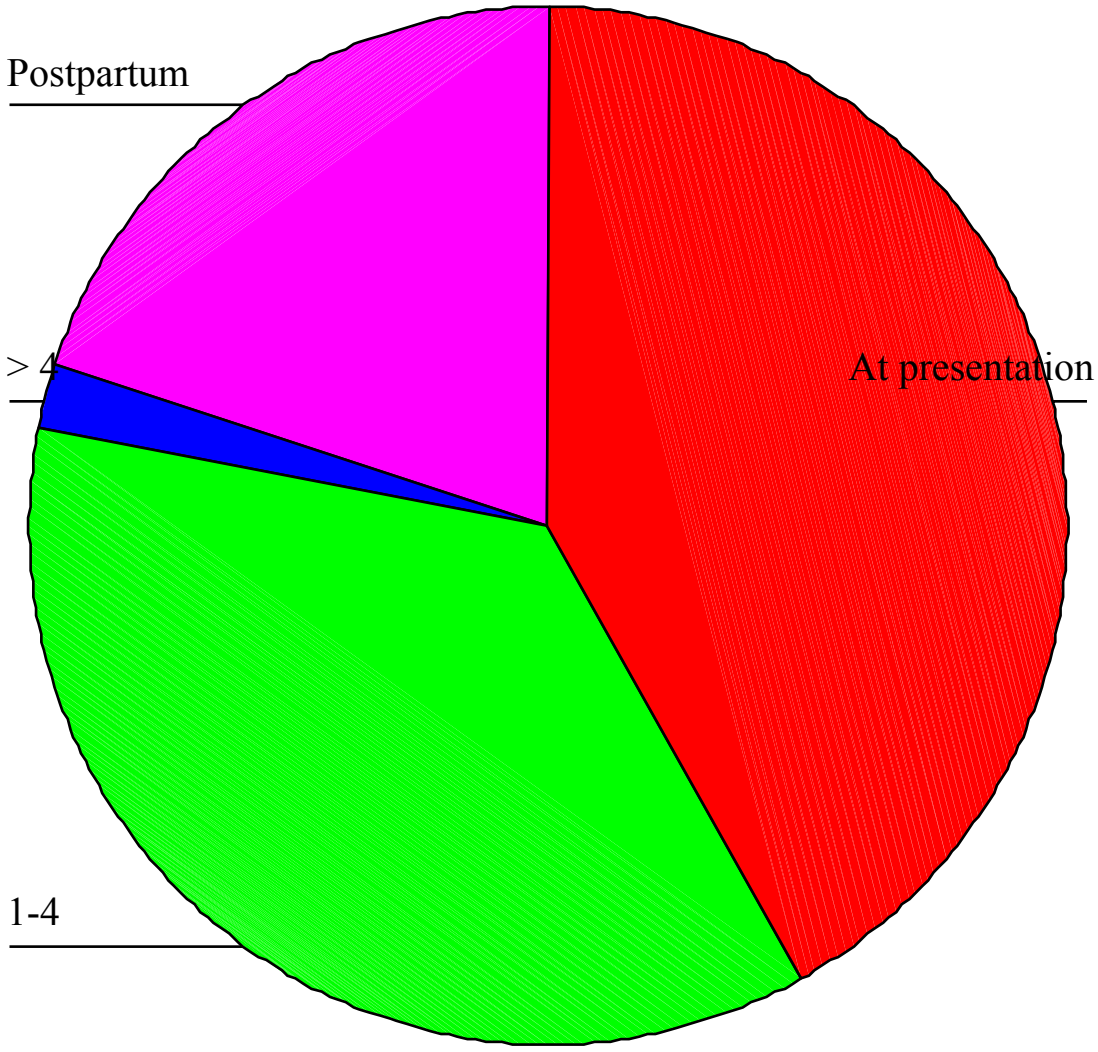

30
20
10
0
Count
20-28
29-36
37-40
Postpartum
GA in weeks
MRI Finding
Normal
Abnormal

DURATION OF HYPERTENSION SINCE DIAGNOSIS

TABLE-5: DURATION OF HYPERTENSION SINCE DIAGNOSIS

	Frequency	Percent
At presentation	21	52.5
1-4 Weeks	18	45.0
> 4 Weeks	1	2.5
Total	40	100.0

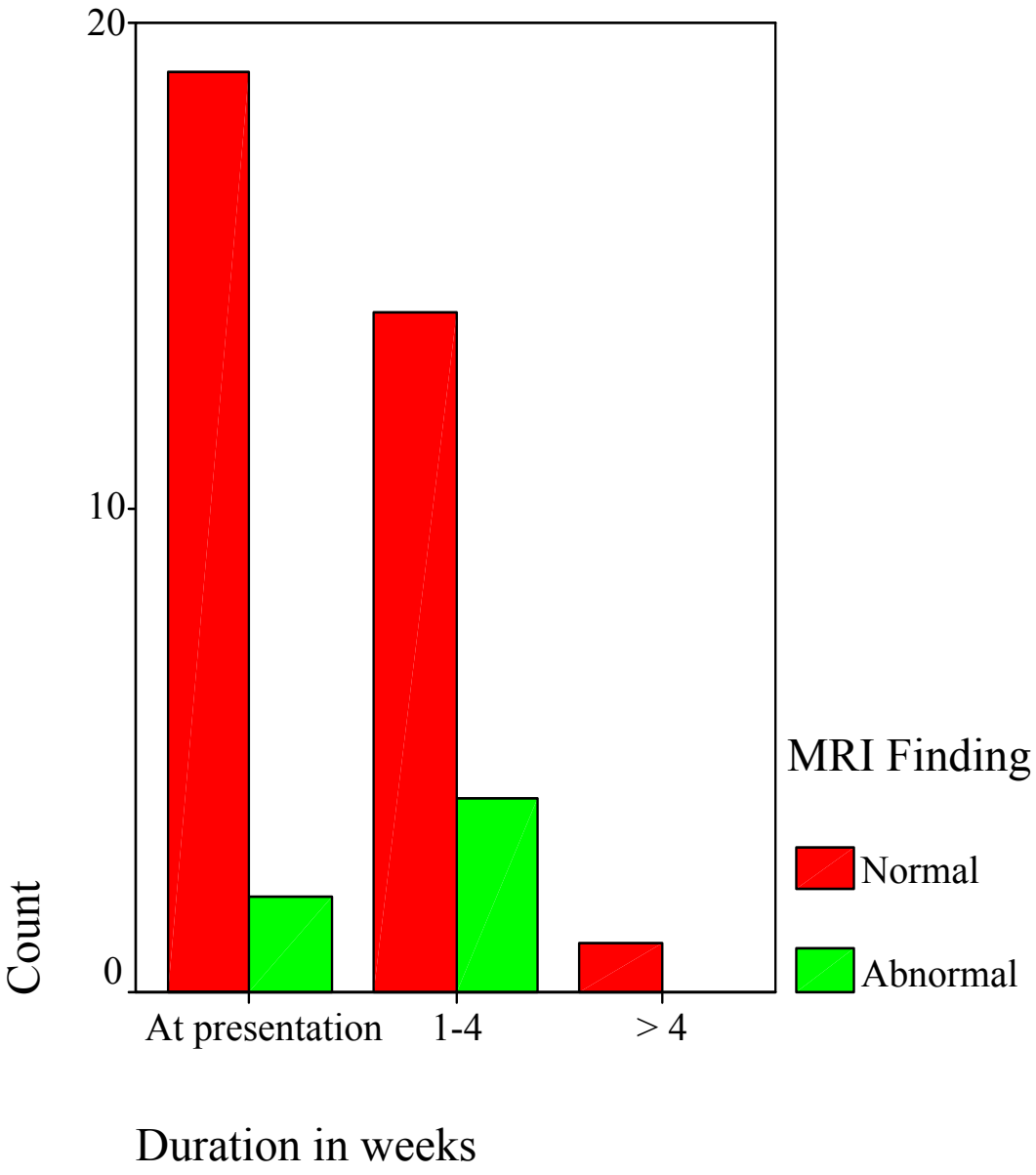
In our study, 52.5% of the patients with eclampsia had hypertension diagnosed only at the time of presentation. Only 2.5% of the patients has hypertension for more than 4 weeks. Rest of the 10% patients are postpartum.


Duration in weeks
Postpartum
> 4
At presentation
1-4

DURATION IN WEEKS*MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
Duration in weeks	At presentation	Count	19	2	.495
		% within Duration in weeks	90.5%	9.5%	
		% within MRI Finding	55.9%	33.3%	
	1-4	Count	14	4	
		% within Duration in weeks	77.8%	22.2%	
		% within MRI Finding	41.2%	66.7%	
	> 4	Count	1	0	
		% within Duration in weeks	100.0%	.0%	
		% within MRI Finding	2.9%	.0%	

Patients who presented with Eclampsia, 1-4 weeks after the onset of hypertension have abnormal MRI finding of 66.7%, p value is 0.495, not significant.

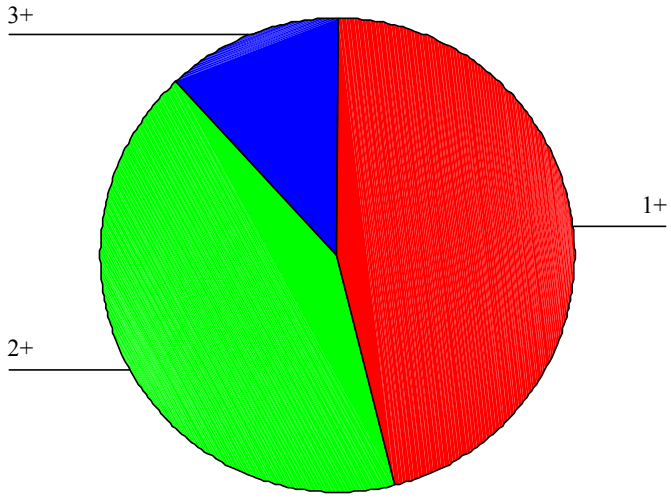

20
10
0
Count
At presentation
1-4
> 4
Duration in weeks
MRI Finding
Normal
Abnormal

PROTEINURIA

TABLE-6: URINE ALBUMIN

URINE ALBUMIN		Frequency	Percent
	1+	6	12
	2+	21	42.0
	3+	6	12.0
	Total	50	100.0

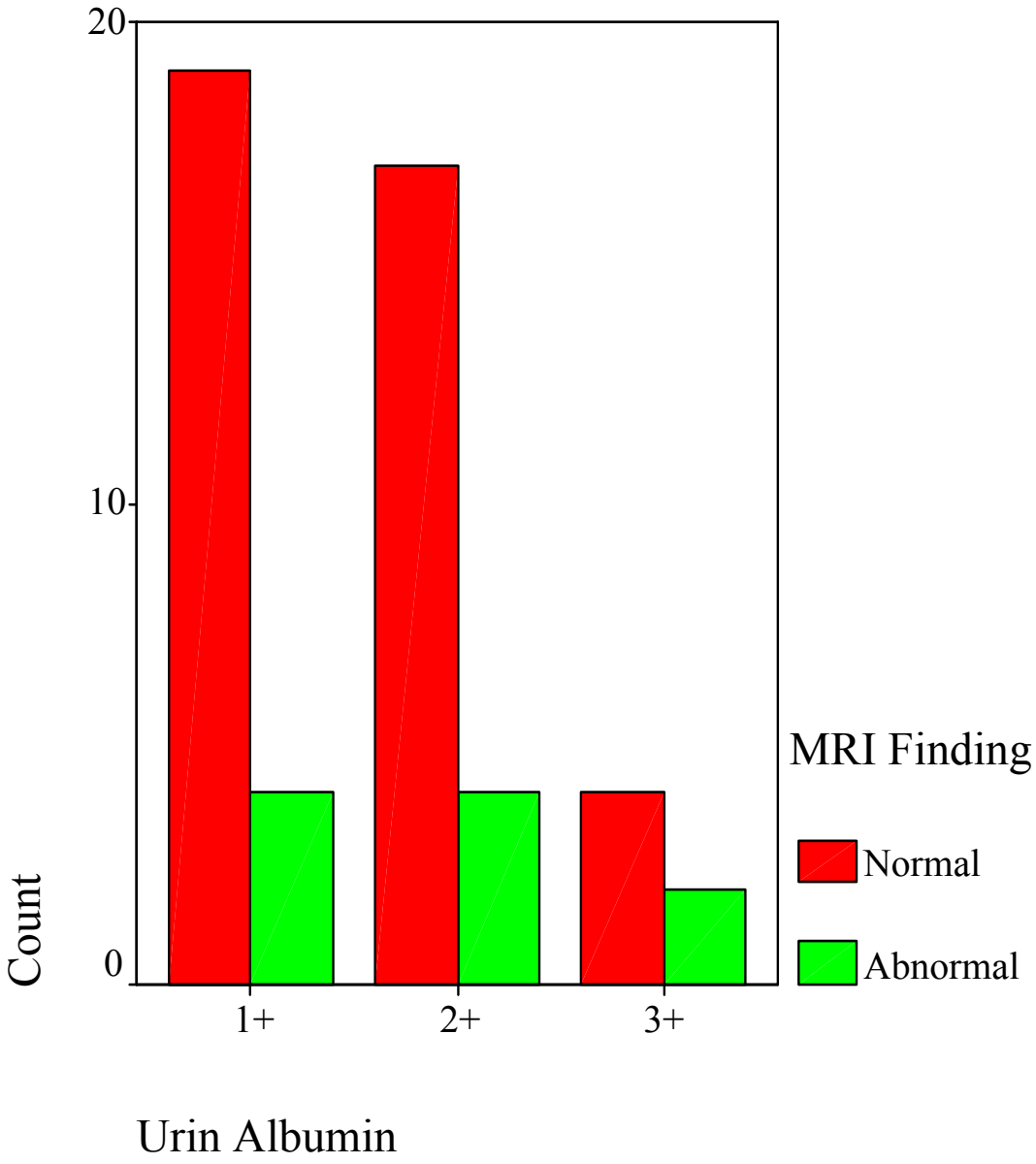
Urin Albumin



PROTEINURIA * MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
Urin Albumin	1+	Count	19	4	.678
		% within Urin Albumin	82.6%	17.4%	
		% within MRI Finding	47.5%	40.0%	
	2+	Count	17	4	
		% within Urin Albumin	81.0%	19.0%	
		% within MRI Finding	42.5%	40.0%	
	3+	Count	4	2	
		% within Urin Albumin	66.7%	33.3%	
		% within MRI Finding	10.0%	20.0%	

In our study group (table 6), 47.5% of the patients with normal MRI findings and 40% of the patients with abnormal MRI findings have proteinuria of 1+. p value is 0.678 which is statistically not significant.

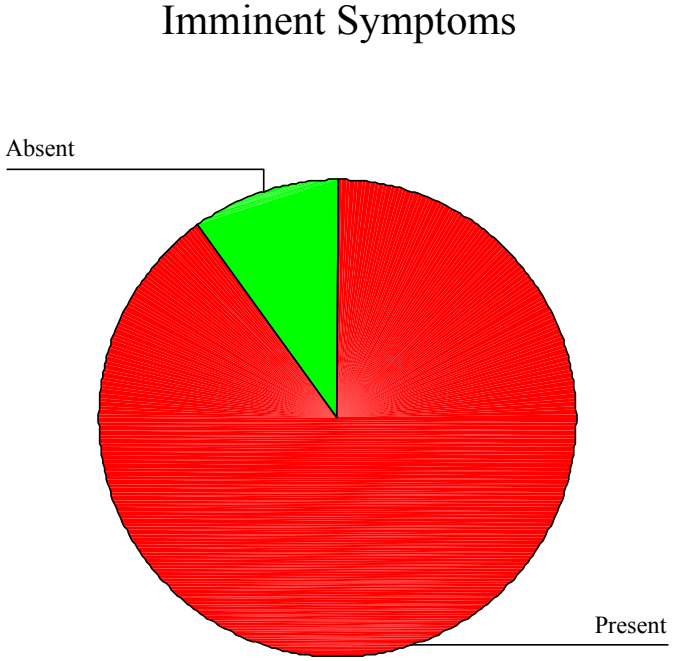

20
10
0
Count
1+
2+
3+
Urin Albumin
MRI Finding
Normal
Abnormal

IMMINENT SYMPTOMS

TABLE -7 : IMMINENT SYMPTOMS

	Frequency	Percent
Present	45	90.0
Absent	5	10.0
Total	50	100.0

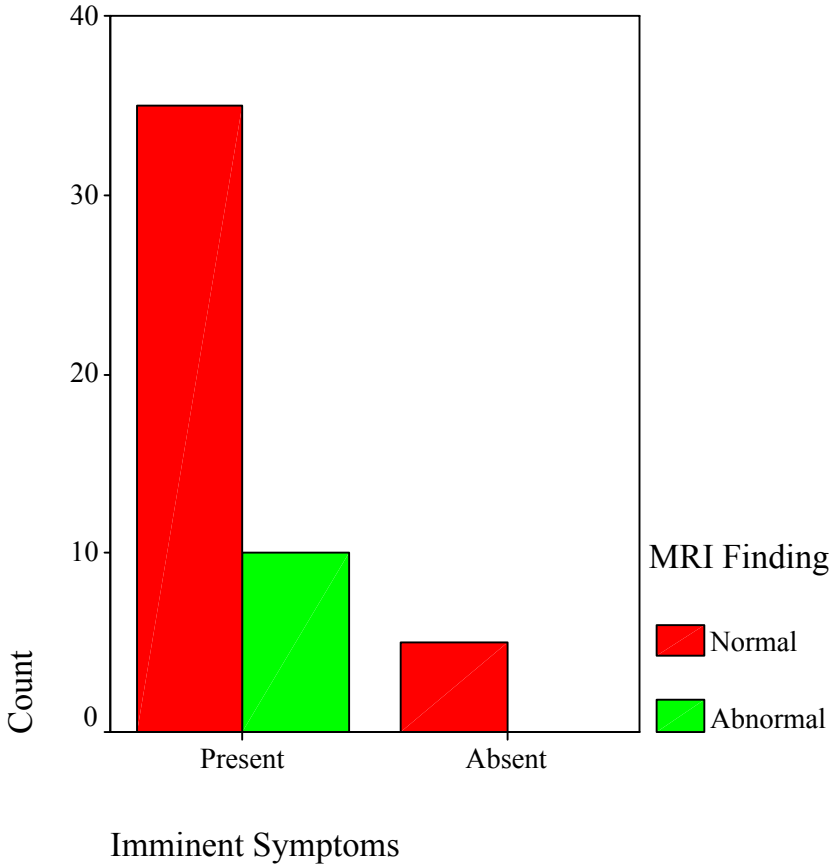
In our study group, 90% of the patients have imminent symptoms, 10% have no imminent symptoms.



IMMINENT SYMPTOMS* MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
Imminent Symptoms	Present	Count	35	10	
		% within Imminent Symptoms	77.8%	22.2%	
		% within MRI Finding	87.5%	100.0%	
	Absent	Count	5	0	
		% within Imminent Symptoms	100.0%	.0%	0.239
		% within MRI Finding	12.5%	.0%	

In our study group, 22.2% of the patients with Imminent symptoms have 100% of abnomal MRI finding. Remaining 77.8% with Imminent symptoms have 87.5% Normal MRI findings. p value = 0.239, which is statistically not significant.

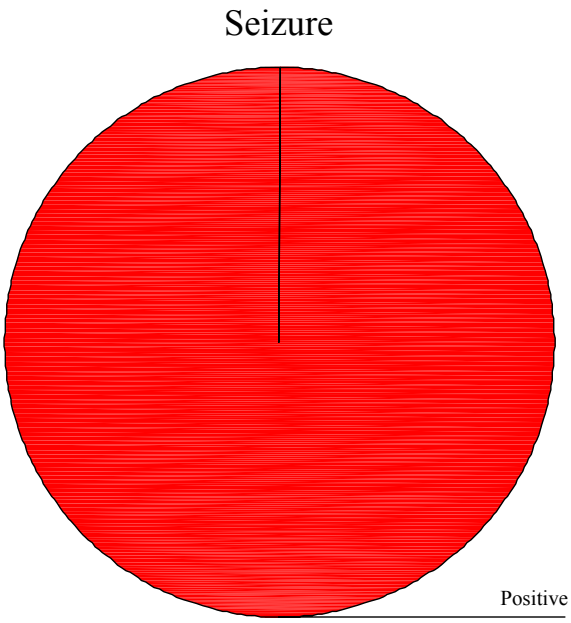


SEIZURE

Table-8: SEIZURE

	Frequency	Percent
Positive	50	100.0

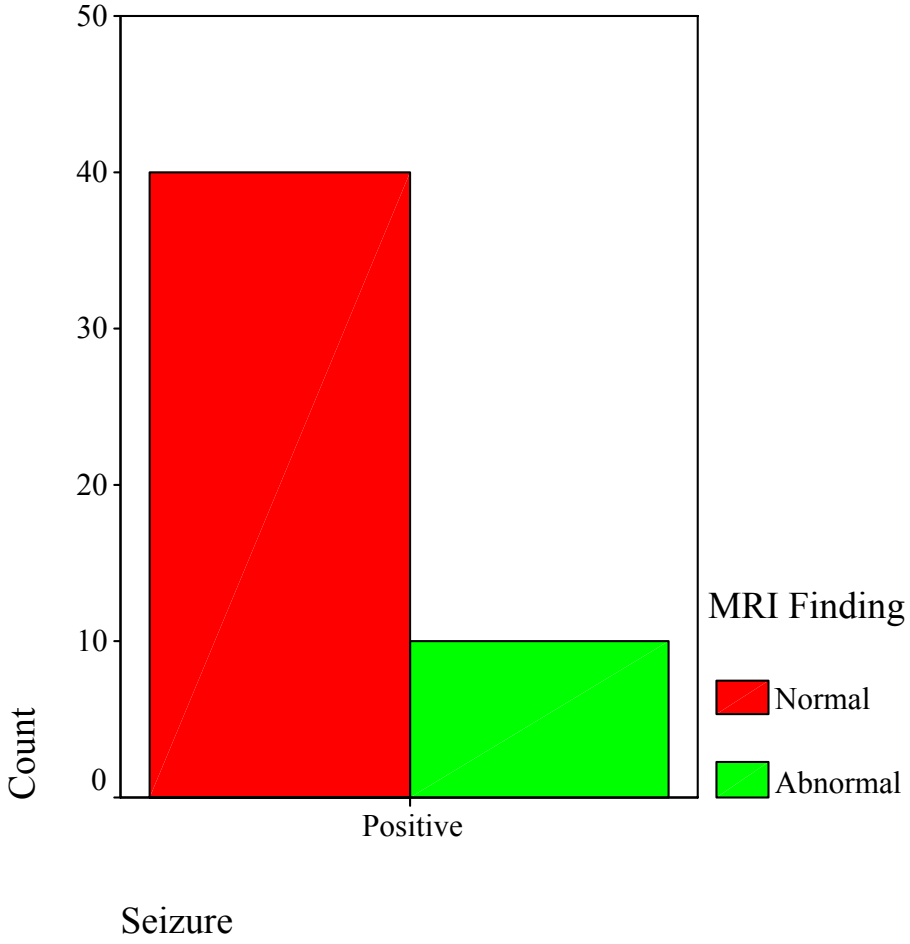
In our study group, all patients had seizures.



SEIZURE*MRI FINDING

			MRI Finding	
			Normal	Abnormal
Seizure	Positive	Count	40	10
		% within Seizure	80.0%	20.0%
		% within MRI Finding	100.0%	100.0%

Only 10% of the Eclampsia patients have abnormal MRI findings.

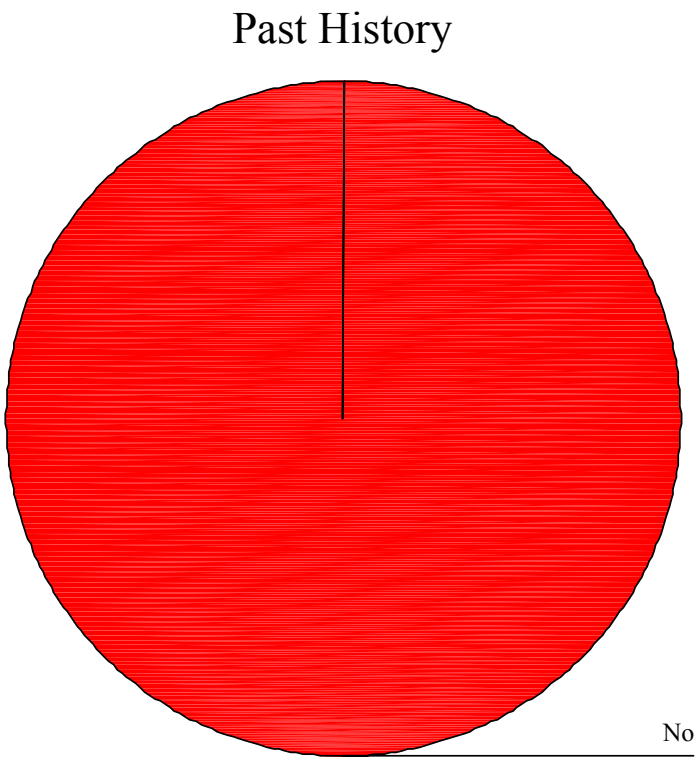

50
40
30
20
10
0
Count
Positive
Seizure
MRI Finding
Normal
Abnormal

PAST HISTORY

Table-9: PAST HISTORY

	FREQUENCY	PERCENT
No	50	100.0

In our study all Eclampsia patients do not have significant past history.



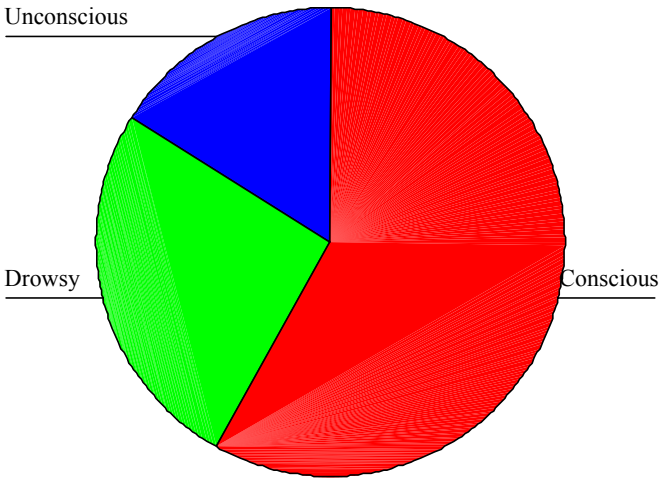
CONSCIOUS LEVEL

Table-10: CONSCIOUS LEVEL

	FREQUENCY	PERCENT
Conscious	29	58.0
Drowsy	13	26.0
Unconscious	8	16.0
Total	50	100.0

Among the patients presenting with Eclampsia, 29 patients were conscious,13 were drowsy and only 8 were unconscious.

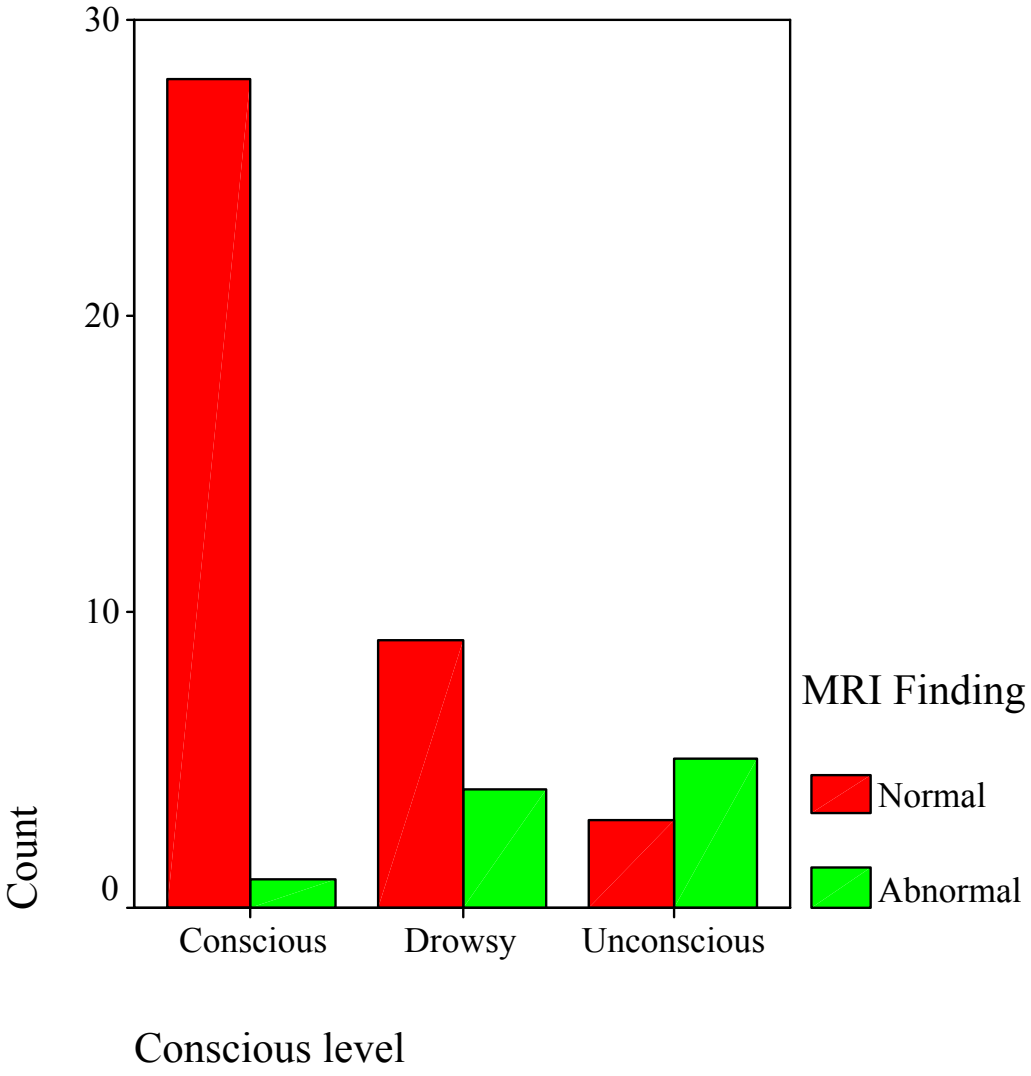
Conscious level



CONSCIOUS LEVEL * MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
Conscious level	Conscious	Count	28	1	
		% within Conscious level	96.6%	3.4%	
		% within MRI Finding	70.0%	10.0%	
	Drowsy	Count	9	4	
		% within Conscious level	69.2%	30.8%	.001**
		% within MRI Finding	22.5%	40.0%	
	Unconscious	Count	3	5	
		% within Conscious level	37.5%	62.5%	
		% within MRI Finding	7.5%	50.0%	

In the study group, 10% of conscious patients,30.8 % of drowsy patients, 50% of unconscious patients have abnormal MRI findings. p value is 0.001 ,which is statistically significant.


30
20
10
0
Count
Conscious
Drowsy
Unconscious
Conscious level
MRI Finding
Normal
Abnormal

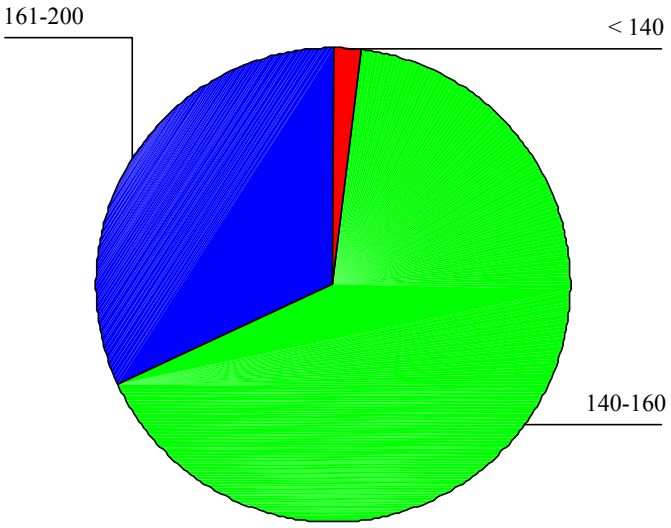
SYSTOLIC BLOOD PRESSURE

Table-11: SBP

	Frequency	Percent
< 140	1	2.0
140-160	33	66.0
161-200	16	32.0
Total	50	100.0

Most of patients in our study group have systolic blood pressure in the range of 140-160 mmHg .only 2% have SBP of <140 mmHg.

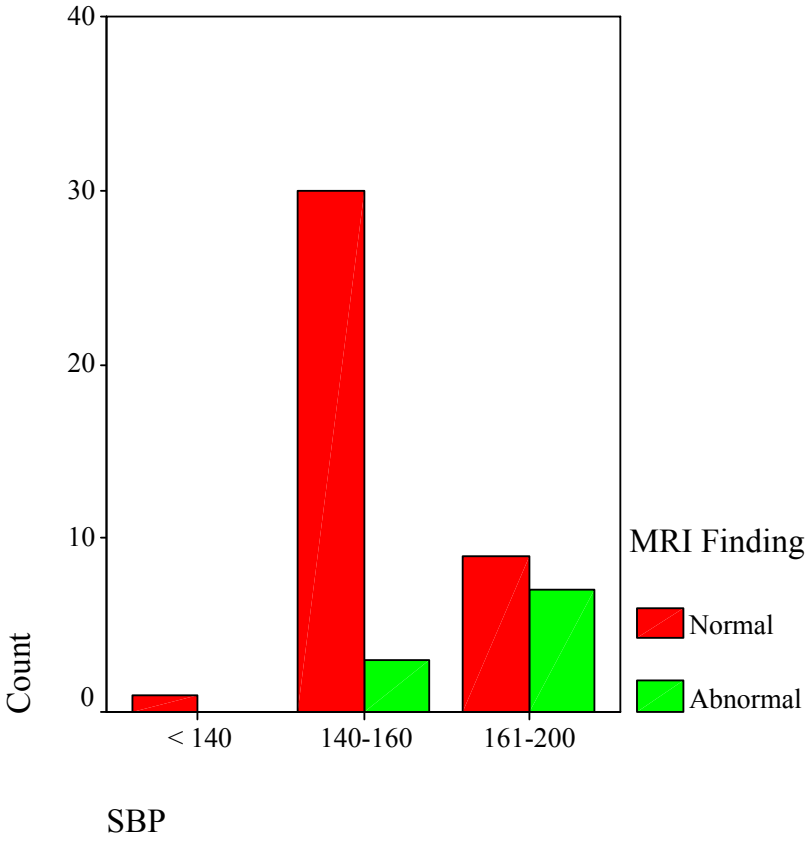
SBP



SBP * MRI Finding

			MRI Finding		
			Normal	Abnormal	P value
SBP	< 140	Count	1	0	
		% within SBP	100.0%	.0%	
		% within MRI Finding	2.5%	.0%	
	140-160	Count	30	3	
		% within SBP	90.9%	9.1%	
		% within MRI Finding	75.0%	30.0%	.015**
	161-200	Count	9	7	
		% within SBP	56.3%	43.8%	
		% within MRI Finding	22.5%	70.0%	

In the study group, 70% of the patients who have abnormal MRI findings have systolic blood pressure in the range of 161-200mmHg. (p value is 0.015**) which is statistically significant.

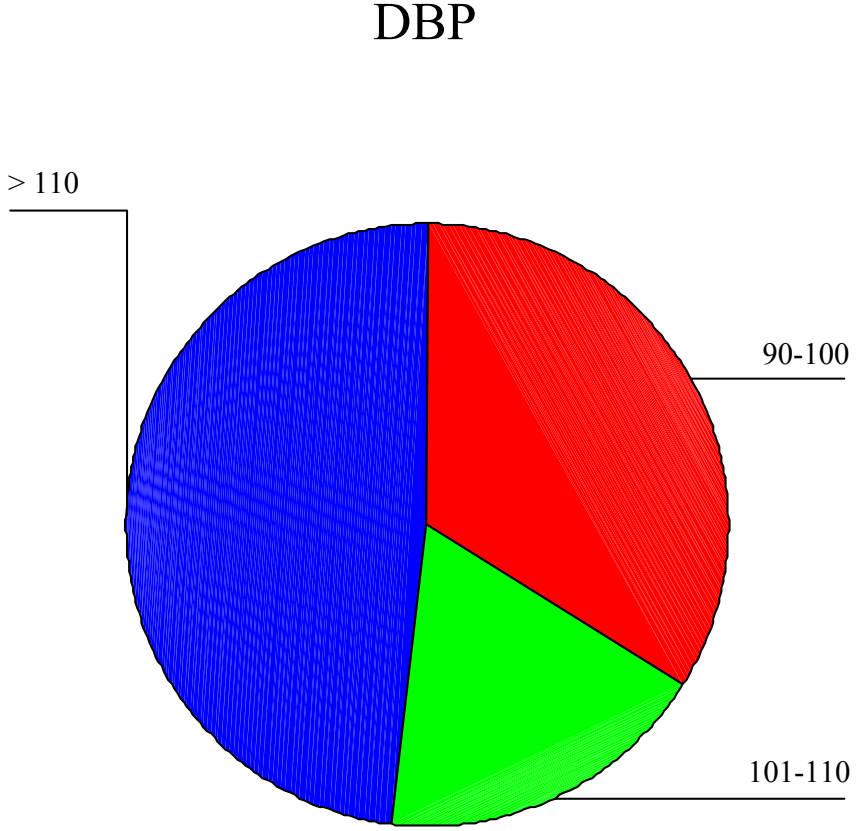

40
30
20
10
0
Count
< 140
140-160
161-200
SBP
MRI Finding
Normal
Abnormal

DIASTOLIC BLOOD PRESSURE

TABLE-12: DBP

	Frequency	Percent
90-100	17	34.0
101-110	9	18.0
> 110	24	48.0
Total	50	100.0

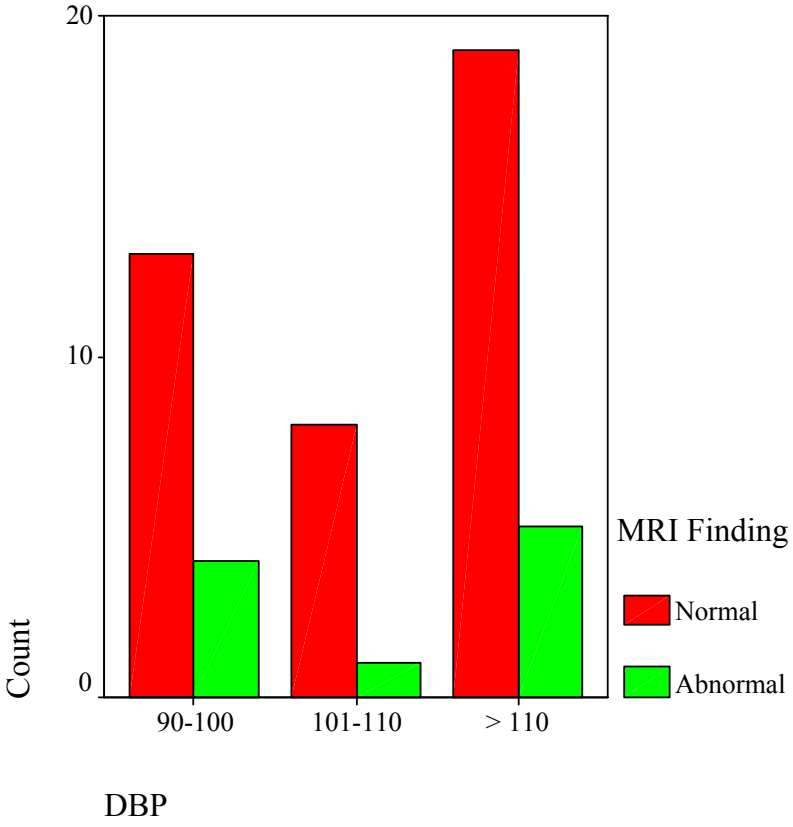
Out of 50 patients in our study group, 48% patients have diastolic blood pressure of more than 110 mmHg. Only 18% have diastolic blood pressure in the range of 101-110 mmHg.



DBP * MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
DBP	90-100	Count	13	4	
		% within DBP	76.5%	23.5%	
		% within MRI Finding	32.5%	40.0%	
	101-110	Count	8	1	
		% within DBP	88.9%	11.1%	
		% within MRI Finding	20.0%	10.0%	.746
	> 110	Count	19	5	
		% within DBP	79.2%	20.8%	
		% within MRI Finding	47.5%	50.0%	

In my study group, 50% of patients with abnormal finding have diastolic blood pressure >110 mmHg. p value is 0.746 which is not significant.



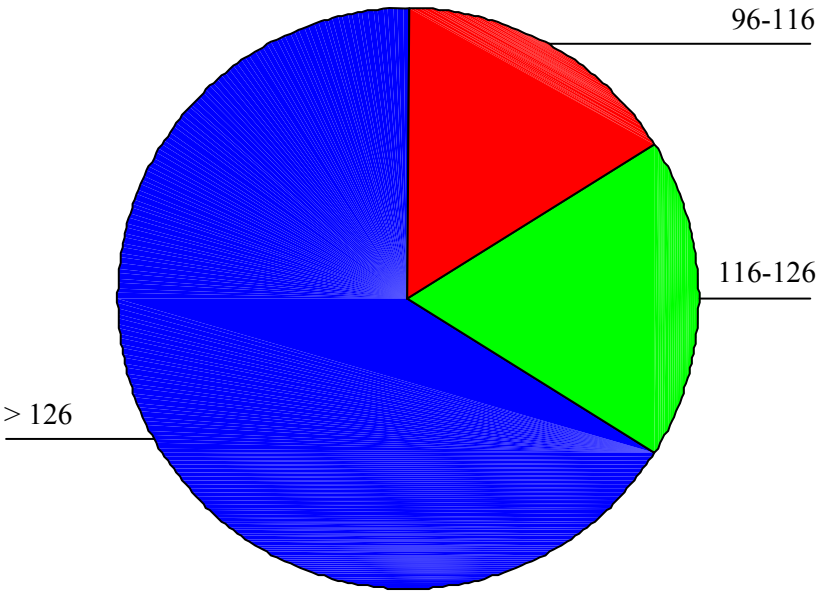
MEAN ARTERIAL PRESSURE

Table-13: MEAN ARTERIAL PRESSURE

	Frequency	Percent
96-116	8	16.0
116-126	9	18.0
> 126	33	66.0
Total	50	100.0

In our study group, 33 patients have their mean arterial pressure >126, where only 8 people have mean arterial pressure in the range of 96-116.

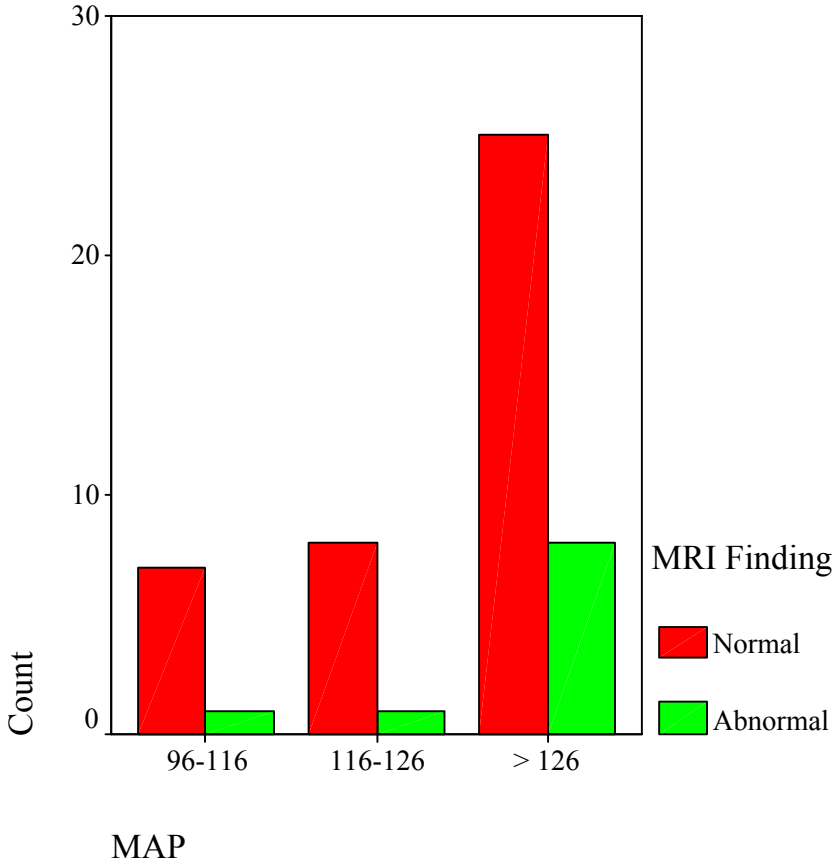
MAP



MAP * MRI Finding

			MRI Finding		
			Normal	Abnormal	P value
MAP	96-116	Count	7	1	
		% within MAP	87.5%	12.5%	
		% within MRI Finding	17.5%	10.0%	
	116-126	Count	8	1	
		% within MAP	88.9%	11.1%	
		% within MRI Finding	20.0%	10.0%	.578
	> 126	Count	25	8	
		% within MAP	75.8%	24.2%	
		% within MRI Finding	62.5%	80.0%	

In my study group, 80% of the patients with abnormal MRI finding have Mean arterial pressure of >126 mmHg. p value is 0.578, not significant.



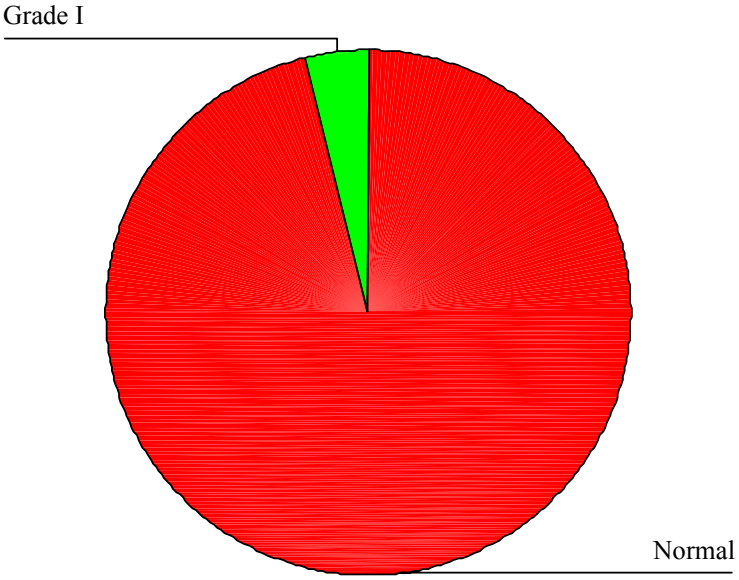
FUNDUS

Table-14: FUNDUS

	Frequency	Percent
Normal	48	96.0
Grade I	2	4.0
Total	50	100.0

Out of 50 patients,48 patients have normal fundus. Only two Patients have hypertensive retinopathy of Grade I.

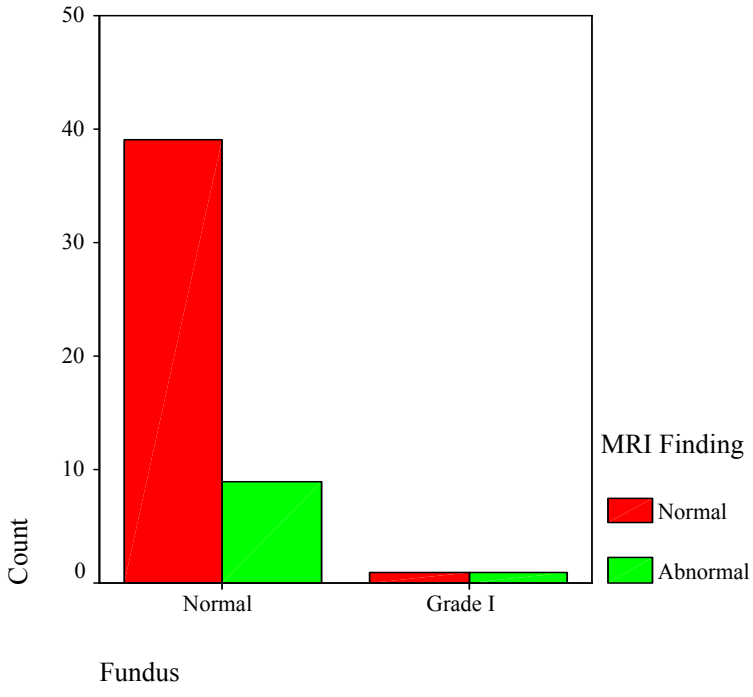
Fundus



FUNDUS * MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
Fundus	Normal	Count	39	9	
		% within Fundus	81.3%	18.8%	
		% within MRI Finding	97.5%	90.0%	
	Grade I	Count	1	1	.279
		% within Fundus	50.0%	50.0%	
		% within MRI Finding	2.5%	10.0%	

In our study group, 90% of the patients with abnormal MRI finding have normal fundus.

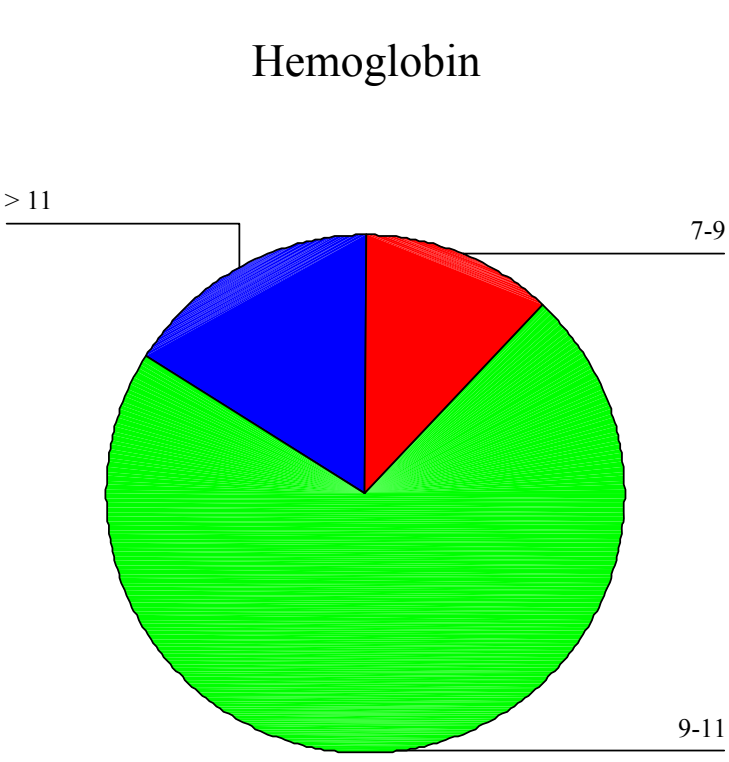


HEMOGLOBIN

TABLE-15: HEMOGLOBIN

	Frequency	Percent
7-9	6	12.0
9-11	36	72.0
> 11	8	16.0
Total	50	100.0

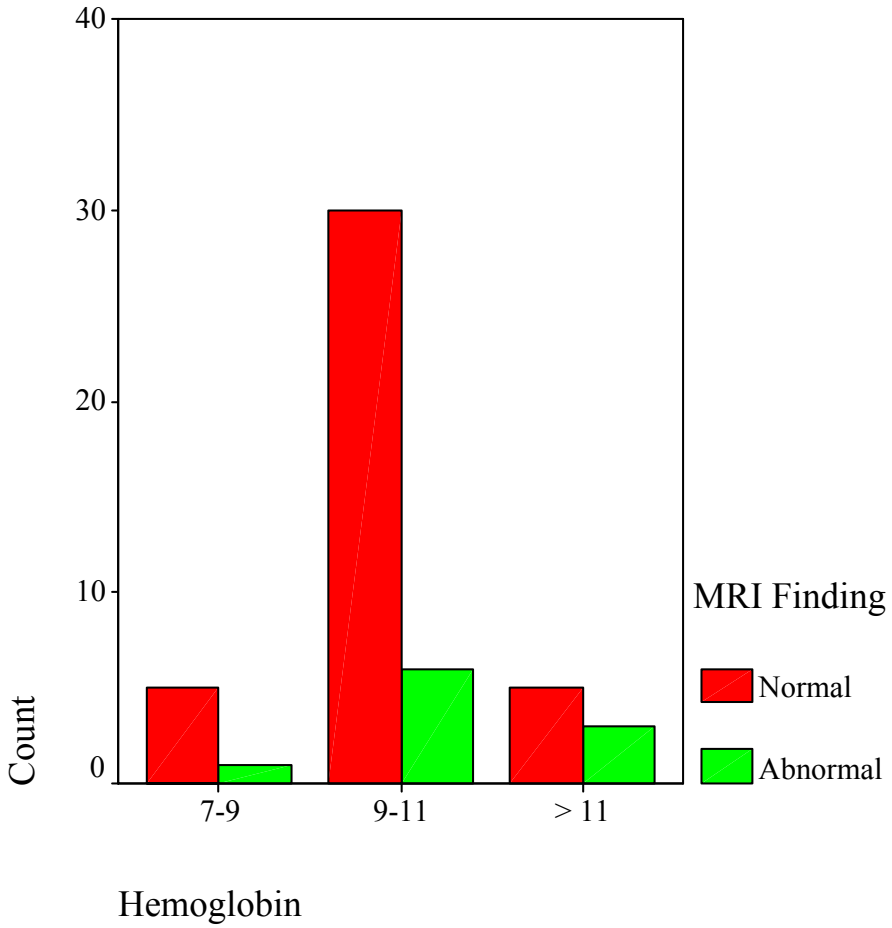
36 patients have Hb% in the range of 9-11g%.6 patients have hemoglobin ranging between 7-9 g%.



HEMOGLOBIN * MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
Hemoglobin	7-9	Count	5	1	
		% within Hemoglobin	83.3%	16.7%	
		% within MRI Finding	12.5%	10.0%	
	9-11	Count	30	6	
		% within Hemoglobin	83.3%	16.7%	
		% within MRI Finding	75.0%	60.0%	.402
	> 11	Count	5	3	
		% within Hemoglobin	62.5%	37.5%	
		% within MRI Finding	12.5%	30.0%	

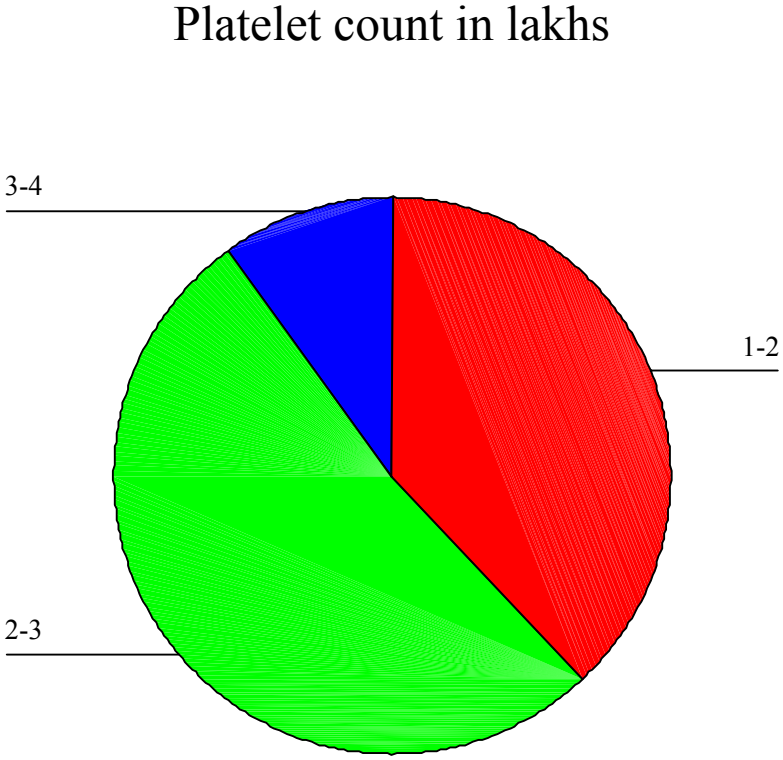
In my study group,60% of the patients having abnormal MRI finding have hemoglobin in the range of 9-11g%.The patients with Hb in the range of 7-9 g% have only 10% of abnormal finding. p value is 0.402 which is not statistically significant.


40
30
20
10
0
Count
7-9
9-11
> 11
Hemoglobin
MRI Finding
Normal
Abnormal

PLATELET COUNT IN LAKHS

Table-16: PLATELET COUNT

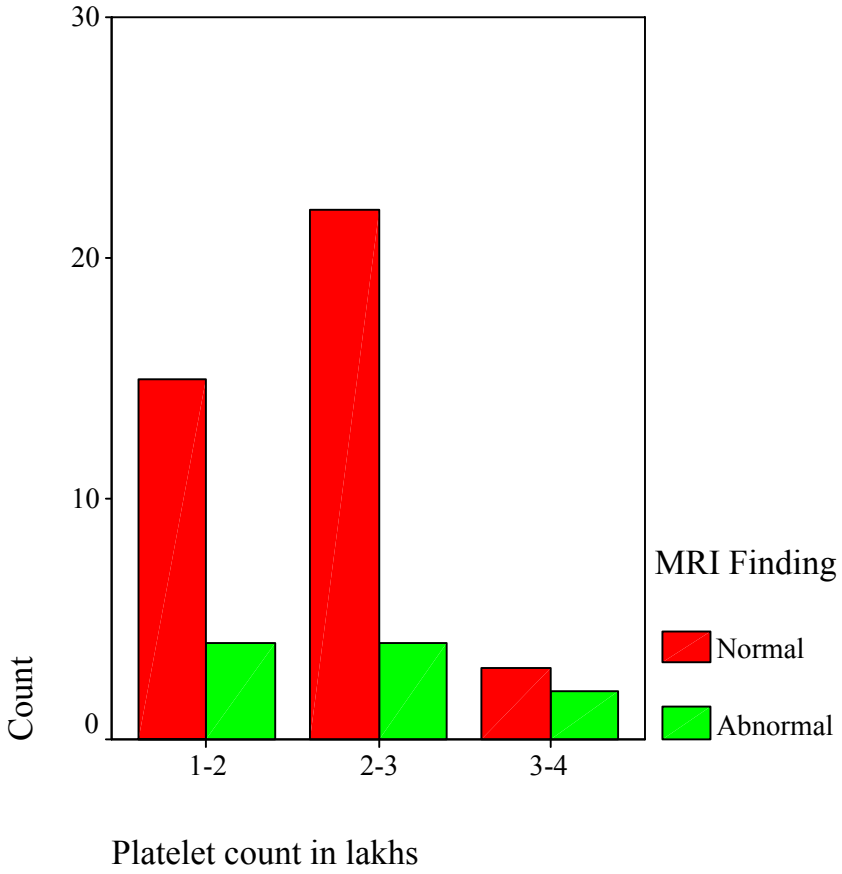
	Frequency	Percent
1-2 L	19	38.0
2-3 L	26	52.0
3-4 L	5	10.0
Total	50	100.0



PLATELET COUNT IN LAKHS * MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
Platelet count in lakhs	1-2	Count	15	4	
		% within Platelet count in lakhs	78.9%	21.1%	
		% within MRI Finding	37.5%	40.0%	
	2-3	Count	22	4	
		% within Platelet count in lakhs	84.6%	15.4%	.447
		% within MRI Finding	55.0%	40.0%	
	3-4	Count	3	2	
		% within Platelet count in lakhs	60.0%	40.0%	
		% within MRI Finding	7.5%	20.0%	

Patients with abnormal MRI finding have platelet count of <3 lakh.p value is 0.447, statistically not significant.

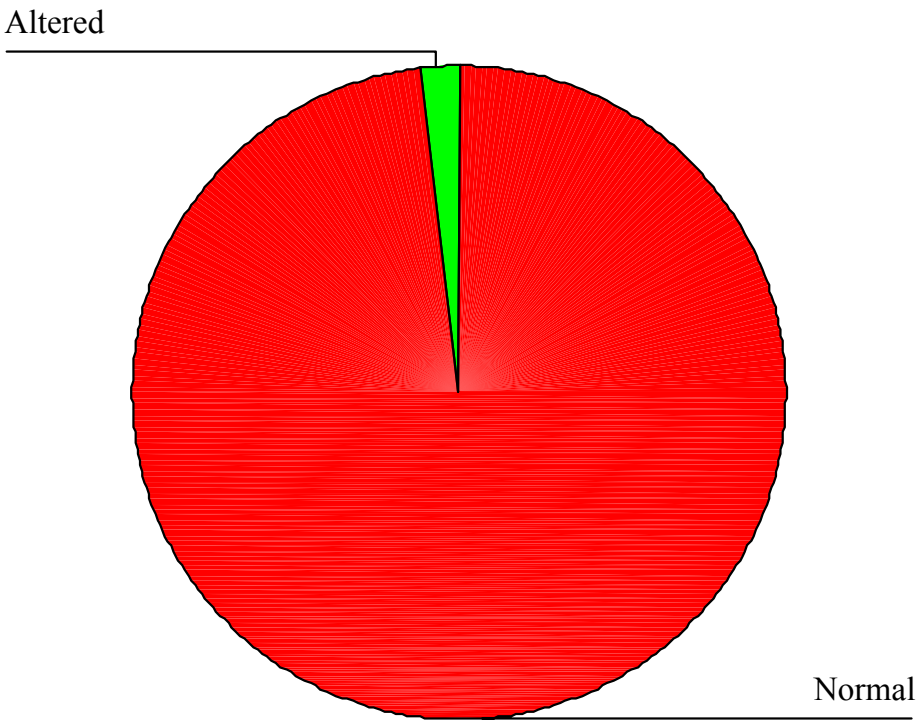

30
20
10
0
Count
1-2
2-3
3-4
Platelet count in lakhs
MRI Finding
Normal
Abnormal

LFT

Table-17: LFT

	Frequency	Percent
Normal	49	98.0
Altered	1	2.0
Total	50	100.0

Out of 50 patients in my study group,only one patient have altered liver function test.

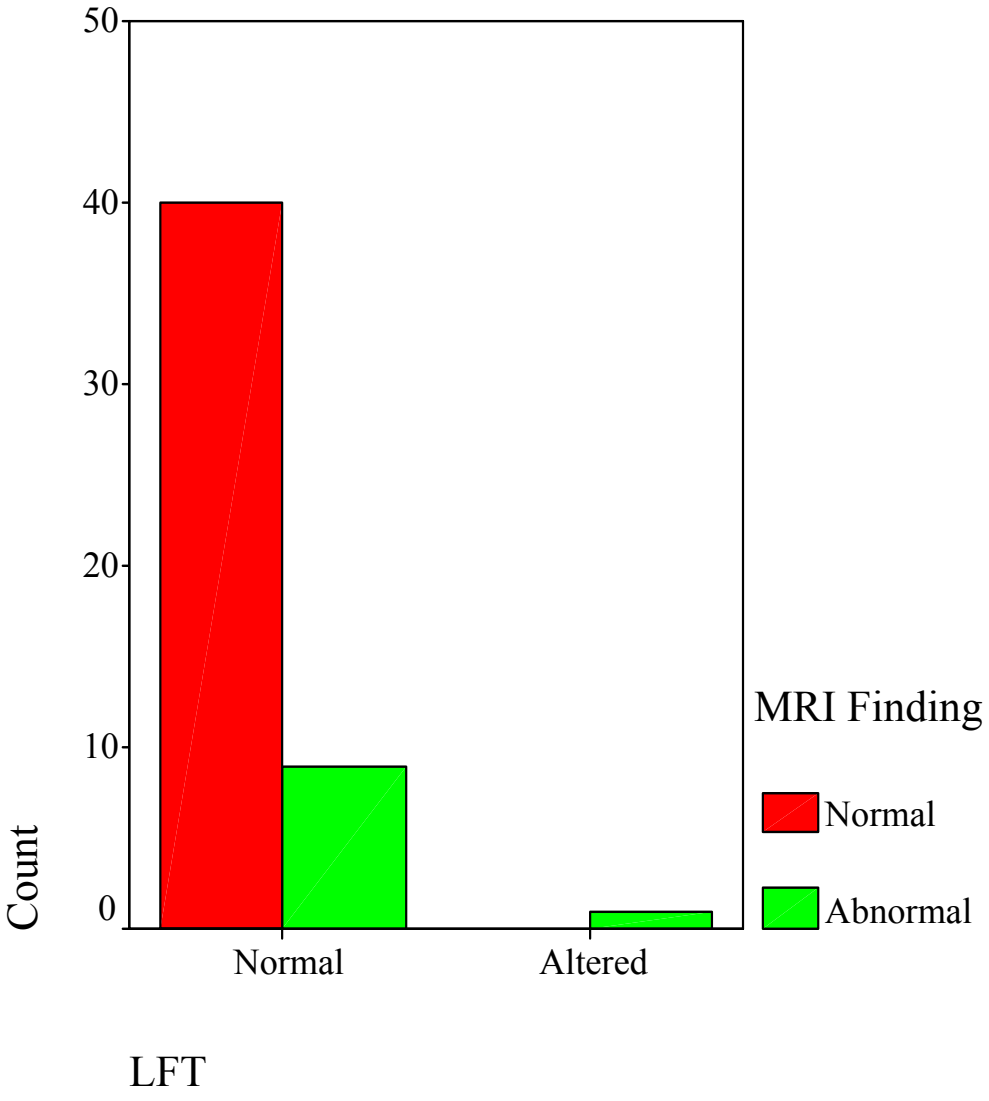


LFT * MRI FINDING

			MRI Finding		
			Normal	Abnormal	P value
LFT	Normal	Count	40	9	
		% within LFT	81.6%	18.4%	
		% within MRI Finding	10.0%	90.0%	
	Altered	Count	0	1	.043**
		% within LFT	.0%	100.0%	
		% within MRI Finding	.0%	100.0%	

In our study group ,the patient with altered liver function test have 100% abnormal MRI finding. Others with normal LFT have 90% have abnormal MRI finding.

p value is 0.043**,significant.

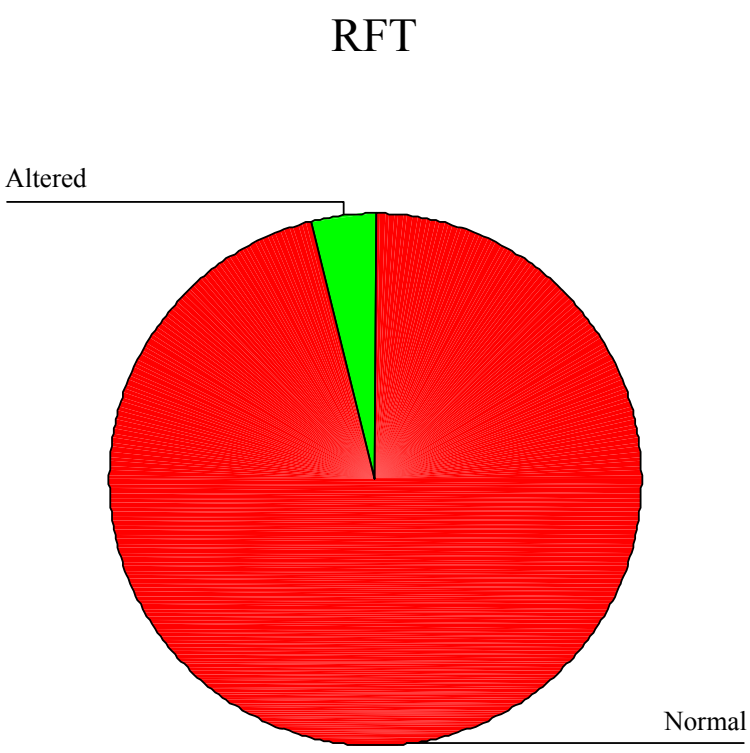

50
40
30
20
10
0
Count
Normal
Altered
LFT
MRI Finding
Normal
Abnormal

RENAL FUNCTION TEST

Table-18:RFT

	Frequency	Percent
Normal	48	96.0
Altered	2	4.0
Total	50	100.0

In our study group, Out of 50 patients, only 2 patients have altered renal function test.

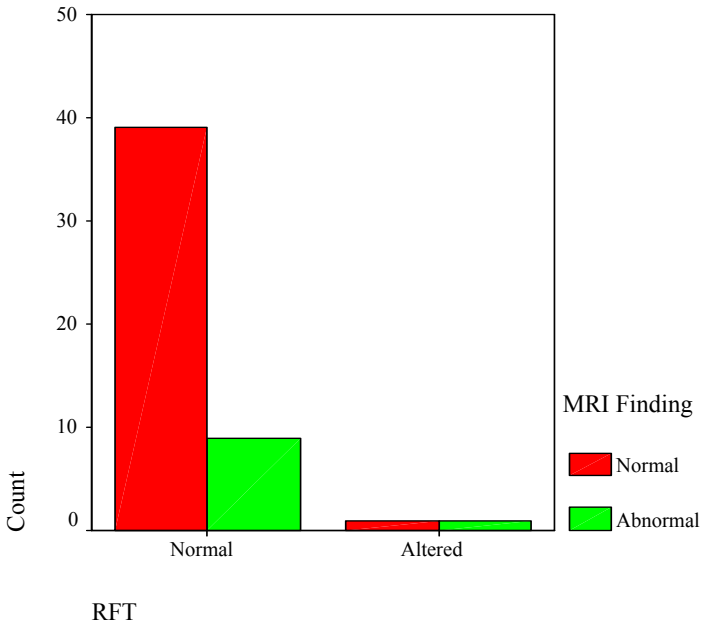


RFT * MRI Finding

			MRI Finding		
			Normal	Abnormal	P value
RFT	Normal	Count	39	9	
		% within RFT	81.3%	18.8%	
		% within MRI Finding	97.5%	90.0%	
	Altered	Count	1	1	
		% within RFT	50.0%	50.0%	.279
		% within MRI Finding	2.5%	10.0%	

Among the patients with altered renal function test,50% have normal and 50% have abnormal MRI finding.

p value is 0.279 ,which is statistically not significant.

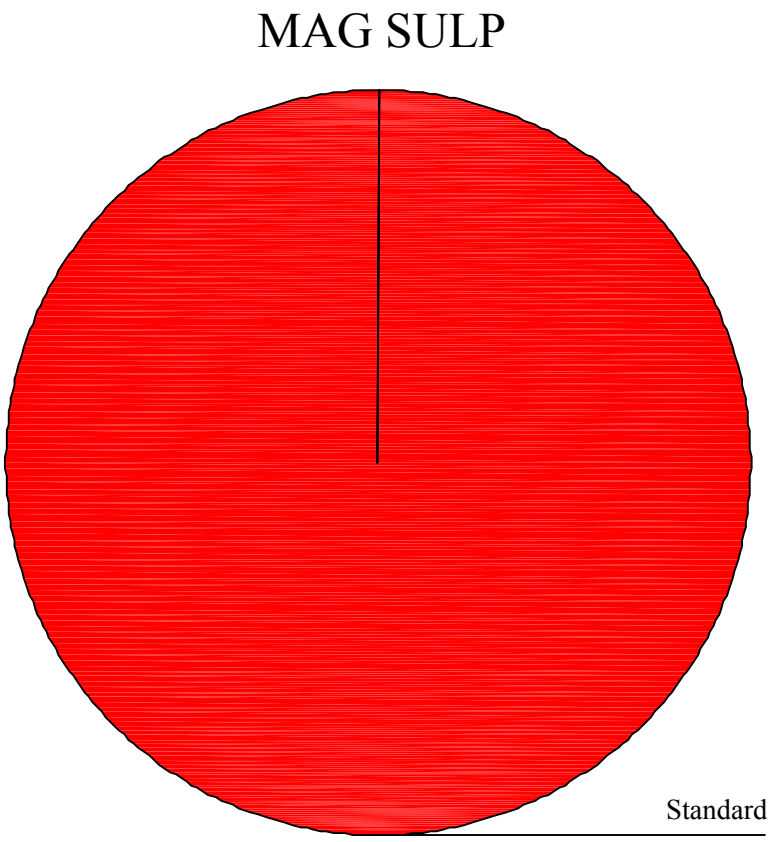


MAGNESIUM SULPHATE

Table-19: MAGNESIUM SULPHATE

	Frequency	Percent
Standard	50	100.0

All Eclamptic patients in our study group were treated with standard dose of Magnesium Sulphate regimen.

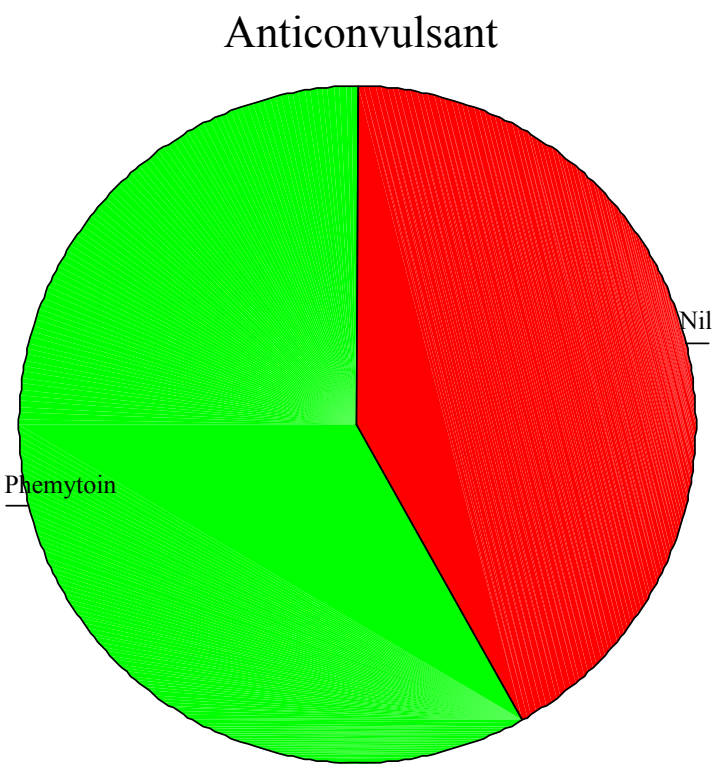


ANTICONVULSANT

Tablet 20- ANTICONVULSANT

	Frequency	Percent
Nil	21	42.0
Phenytoin	29	58.0
Total	50	100.0

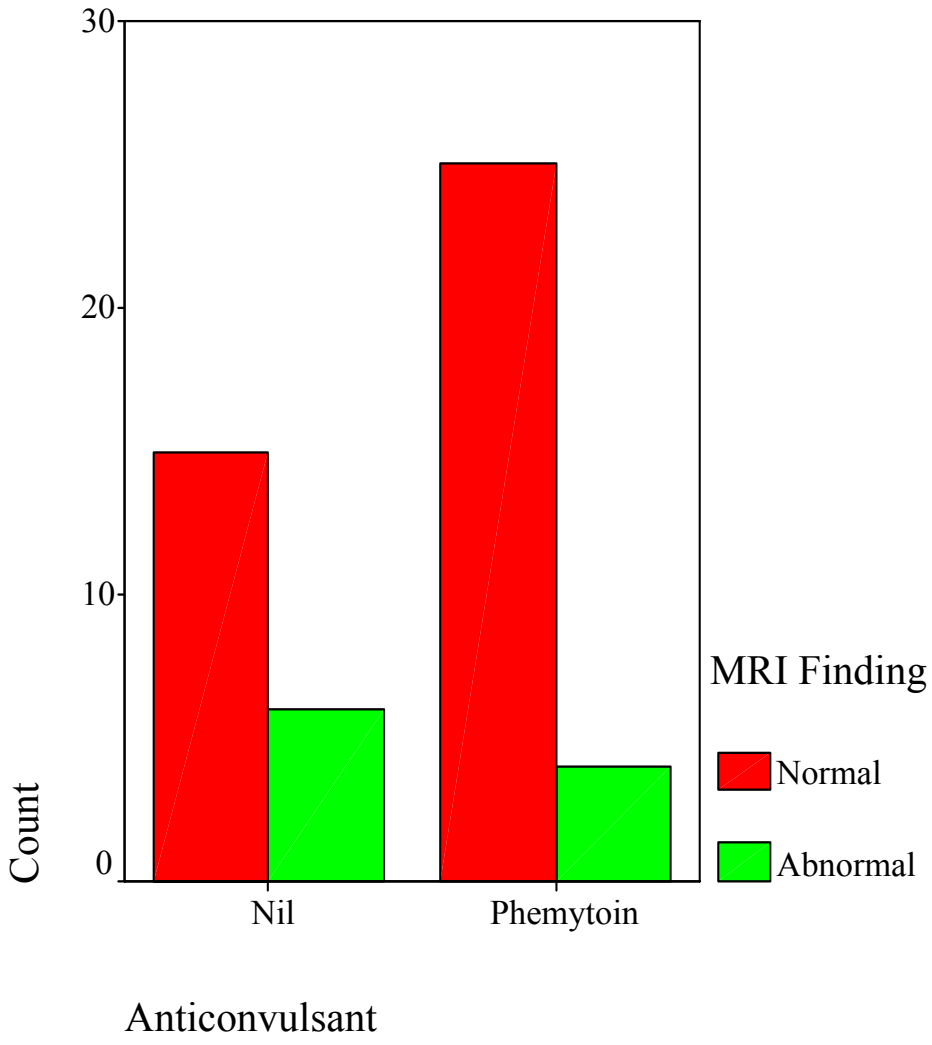
In our study group only 29 Eclampsia patients were treated with Phenytoin in addition to Magnesium sulphate. Remaining 21 patients are treated only with Magnesium sulphate.



Anticonvulsant * MRI Finding

			MRI Finding		
			Normal	Abnormal	P value
Anticonvulsant	Nil	Count	15	6	
		% within Anticonvulsant	71.4%	28.6%	
		% within MRI Finding	37.5%	60.0%	
	Phenytoin	Count	25	4	.176
		% within Anticonvulsant	86.2%	13.8%	
		% within MRI Finding	62.5%	40.0%	

In our study group, among the patients treated with IV Phenytoin ,62.5% have normal MRI finding. Those who are not treated , have 37.5% of normal finding and 60% abnormal finding .p value is 0.176,which is not Significant.


30
20
10
0
Count
Nil
Phemytoin
Anticonvulsant
MRI Finding
Normal
Abnormal

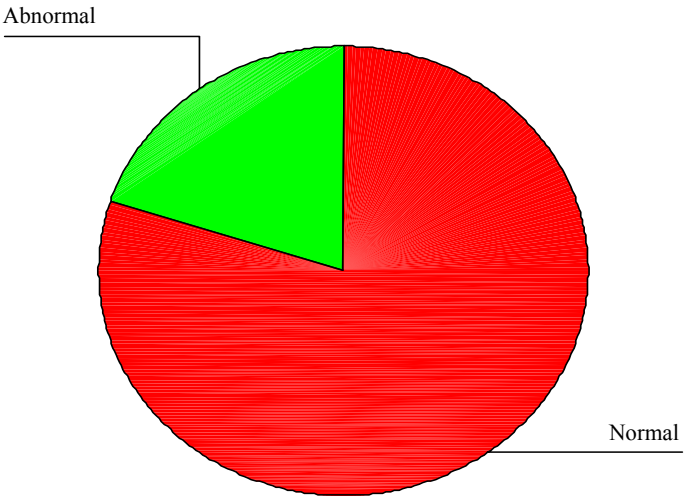
MRI FINDING

TABLE-21: MRI FINDING

		Frequency	Percent
Valid	Normal	40	80.0
	Abnormal	10	20.0
	Total	50	100.0

In our study group,80% have normal MRI finding,20% have abnormal MRI finding.

MRI Finding



MRI FINDING

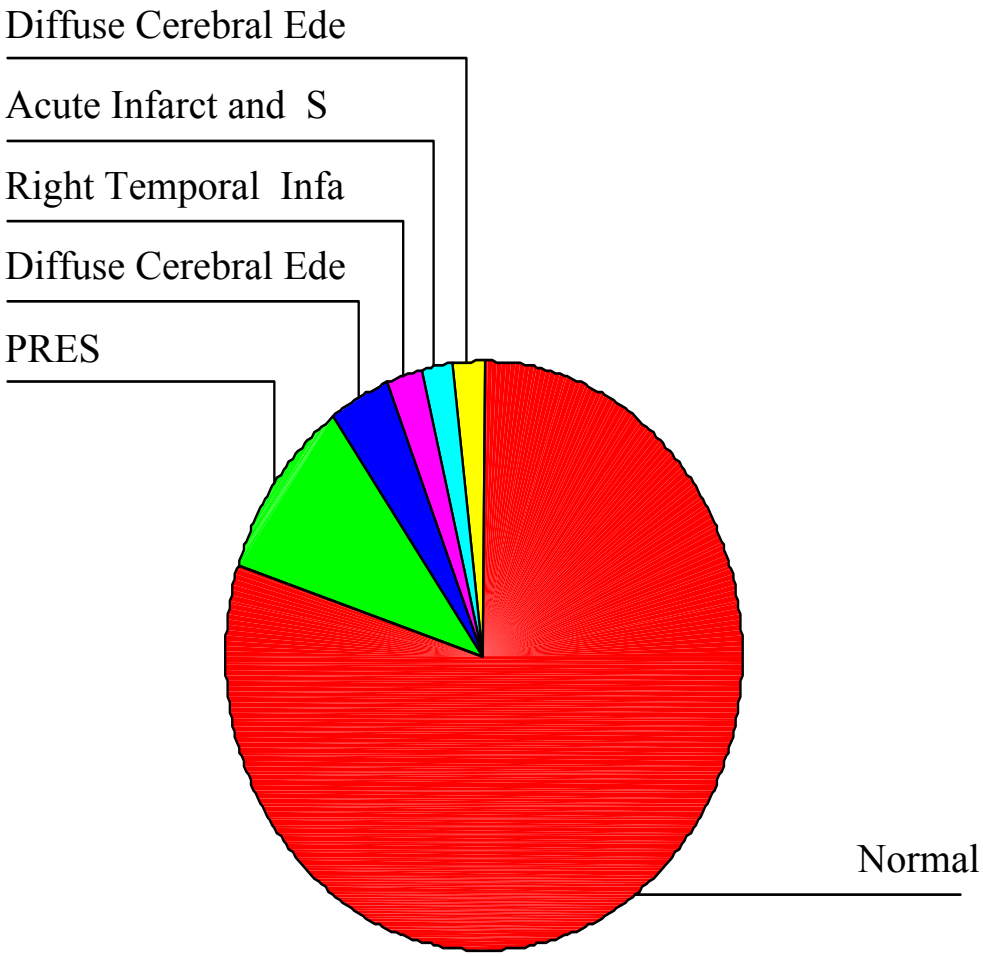
Table-22: MRI FINDING

	Frequency	Percent
Normal	40	80.0
PRES	5	10.0
Diffuse Cerebral Edema	2	4.0
Right Temporal Infarct	1	2.0
Acute Infarct and Subarachnoid Hemmorhage	1	2.0
Diffuse Cerebral Edema + PRES	1	2.0
Total	50	100.0

Out of 50 Eclampsia patients in our study group,40 patients have normal MRI finding.Out of 50, 5 patients are diagnosed as having Posterior Reversible Encephalopathy syndrome.2 patients have diffuse cerebral edema ,1 patient has Right temporal infarct ,1 patient has combination of both Acute infarct and Subarachnoid Hemmorrhage.

1 patient has both diffuse cerebral edema and PRES.

MRI Finding


Diffuse Cerebral Ede
Acute Infarct and S
Right Temporal Infa
Diffuse Cerebral Ede
PRES
Normal

DISCUSSION

In this study 50 women with eclampsia were selected according to the inclusion exclusion criteria and Magnetic Resonance and Imaging was done .The reports were analysed and studied.

In our study (Table-1), the most common age group was 21-25 yrs.58% of the patients falls between 21-25 yrs of age.

18 % of the patients are >25 yrs of age.24% are below 20yrs of age are .50% of the patients between the age group of 21-25 yrs have abnormal MRI finding. p value is 0.543 which is statistically not significant.

In our study (Table-2) majority of the patients are multigravida, which constitutes 60%. 80% of the patients having abnormal MRI findings are multigravida.

Similarly in a study by Milliez et al majority of the patients are multigravida.

In our study group (Table-3),54% of the patients have normal BMI (19.8-26) .14% are obese with BMI>29.4%.90% of the patients with normal BMI have abnormal MRI finding. p value is 0.036 which is statistically significant.

In our study group (Table-4), majority of the patients are antepartum at the time of presentation.20% are postpartum. Majority of the eclampsia patients in our study are between 29-36 weeks of gestational age. p value is 0.003** which is statistically significant.

In our study (table 5) majority had hypertension diagnosed at the time of presentation that is 52.5% of the patients had hypertension diagnosed only at the time of presentation with eclampsia.66.7%of the patients

having abnormal MRI findings presented with eclampsia 1-4 weeks after the onset of hypertension.

In a study made by chakravarthy et al all the patients except one did not have any evidence of pre eclampsia till the last clinical visit.

From this we conclude that majority of the eclampsia patients are diagnosed to have hypertension at the time of presentation.

In our study group (table 6), 47.5% of the patients with normal MRI findings and 40% of the patients with abnormal MRI findings have proteinuria of 1+. p value is 0.678 which is statistically not significant.

In our study group (table 7), majority of the patients (90%) had imminent symptoms, 10% had no imminent symptoms, p value is 0.239 which is not significant

In our study group (table 7) all the patients had seizures.

In our study group (table 9) all the patients didn't have significant positive history.

In our study group (table 10) 58 % of the patients who are conscious had 10% of abnormal MRI findings. 26 % of the patients who are drowsy had 40% of abnormal MRI findings. Majority of the patients who are having abnormal MRI findings are unconscious (60%). P value is 0.001 which is statistically significant. Among the patients with normal MRI finding,62.5% were unconscious,37.5% were conscious.

In our study group (table-11),Patients with systolic blood pressure ranging from 161-200mHg,70% have abnormal MRI finding,22.5% have normal MRI finding. The patients with systolic blood pressure<160mmHg,had majority of normal MRI findings than

abnormal.p value is 0.015, which is significant.Hence high systolic blood pressures have significant cerebral pathology.

In our study group (table 12), the patients with diastolic blood pressures>110mmHg have 50% abnormal MRI finding and 47.5% of abnormal finding.p value is 0.746 which is statistically not significant.

In our study group (table 13),the patients with Mean arterial pressure>126 mmHg,have 80% of abnormal MRI findings and 62.5% of normal MRI findings.p value is 0.578,which is not significant.

In a study done by chakravarthy et al,all patients had systolic BP more than 140mmHg and diastolic >100mmHg.

In our study group (table 14) the patients with the Grade 1 hypertensive retinopathy have only 10% of abnormal MRI findings. p value is 0.279 which is statistically not significant.

In a study done by chakravarthy et al almost all the patients had fundus changes.

In a study group (table 15) majority of the patients were mildly anaemic that is Hb% between 9-11 gm%. These patients have 60 % of abnormal MRI findings. Among the patients with Hb 7-9 gm% only 10% had abnormal MRI findings. p value is 0.402, which is not significant.

In our study group (table 16) patients with more than 3 lakhs of platelet count have only 20 % of abnormal MRI findings. Those with less than 3 lakhs of platelet count had 80 % of abnormal MRI finding. p value is 0.447 which is not significant.

In a study done by Hira B and Moodley 70% had thrombocytopenia.

In our study group (table 17) patients with altered liver function test had 100% of abnormal MRI finding. p value is 0.043 which is statistically significant.

In a study done by Hira B and Moodley 7% of the patients had abnormal liver function test .

In our study group (table 18) among the patients with altered renal function test 50% had normal MRI findings. p value is 0.279 which is not significant.

Zhu XW done a study in which patients with renal impairment were found to be more susceptible to cerebral lesions

In our study group (table 19) all the patients were treated with standard dose of magnesium sulphate regimen.

But in the study done by chakravarthy et al, none of the patients were treated by Magnesium Sulphate. All the patients in the study group of Chakravarthy et al were managed by neurologists and not by obstetricians.

In our study group (table 20) those patients who were treated with IV phenytoin in addition to magnesium sulphate had 62.5 % of normal MRI findings. Those who were not treated with IV Phenytoin had 60 % of abnormal MRI finding. p value is 0.176 which is not statistically significant.

In a study done by Chakravarthy et al all patients were treated with IV phenytoin.

In our study group,(table 21) Out of 50 Eclampsia patients ,40 patients had normal MRI finding.5 patients are diagnosed as having Posterior Reversible Encephalopathy syndrome.2 patients had diffuse

cerebral edema ,1 patient had Right temporal infarct ,1 patient had combination of both Acute infarct and Subarachnoid Hemmorrhage.1 patient had both diffuse cerebral edema and PRES.

In the study done by chakravarthy et al all patients(n=8)had cerebral odema,3 patients had cerebral hemmorhage(37%)

In a study done by Milliez et al out of 18 patients with positive MRI findings,3 had cerebrovascular damage,6 had cerebral oedema,9 had accentuated cortical sulci leading to the diagnosis of communicating hydrocephalus with cortical atrophy.

In a study done by Moodley et al (1993),the most common finding was cerebral edema.

In our study group (table 22), the most common finding is Posterior Reversible Encephalopathy Syndrome, next is the diffuse cerebral oedema.

In our study group,the most common area affected by PRES and hemmorhage is parieto occipital region as we discussed earlier ,posterior circulation is more prone to acute hypertension as it lacks perivascular sympathetic nerves .

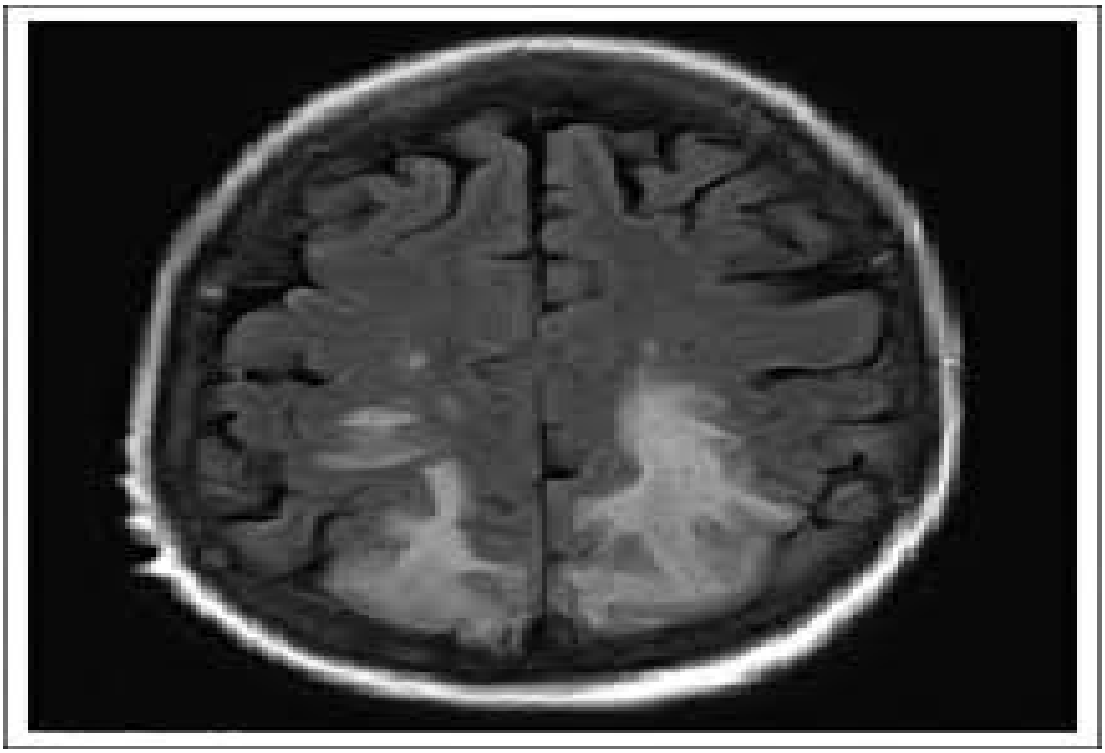
In a study done by chakravarthy et al, the most affected area was occipital region. Brainstem or Basal ganglia is free of lesion.

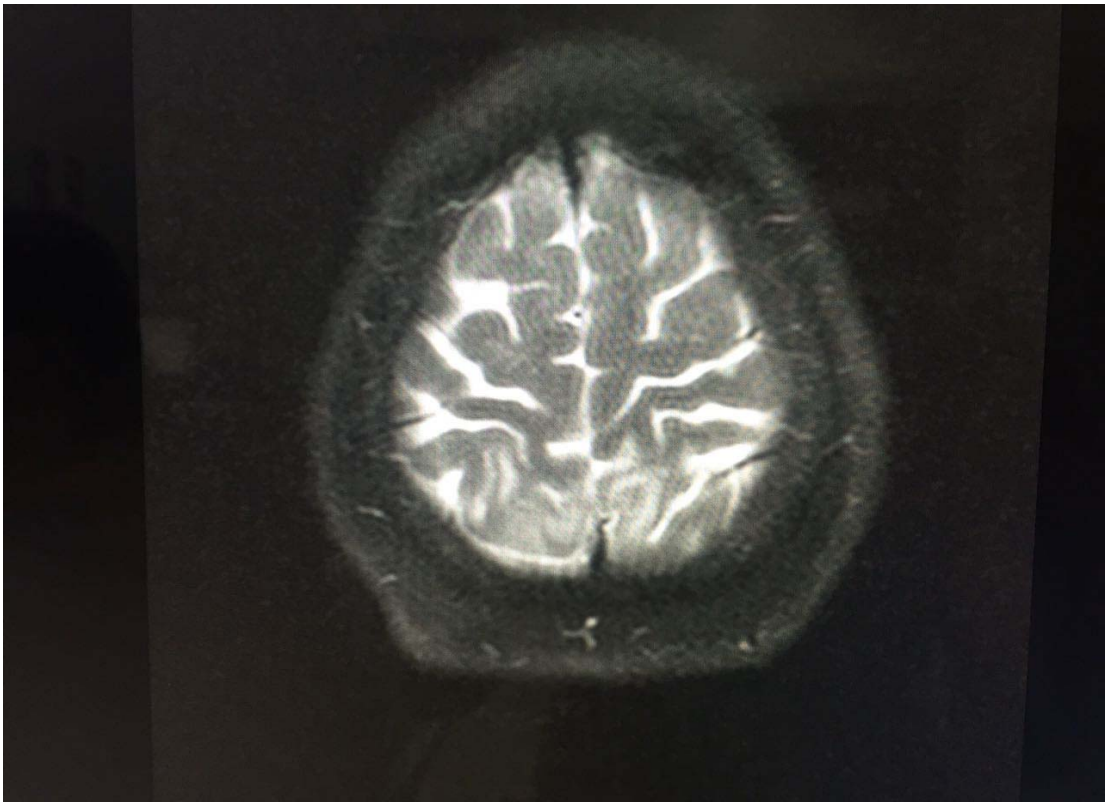
Zhu XW in his study, stated that cerebral lesions were found in cortical and subcortical areas of bilateral parietal

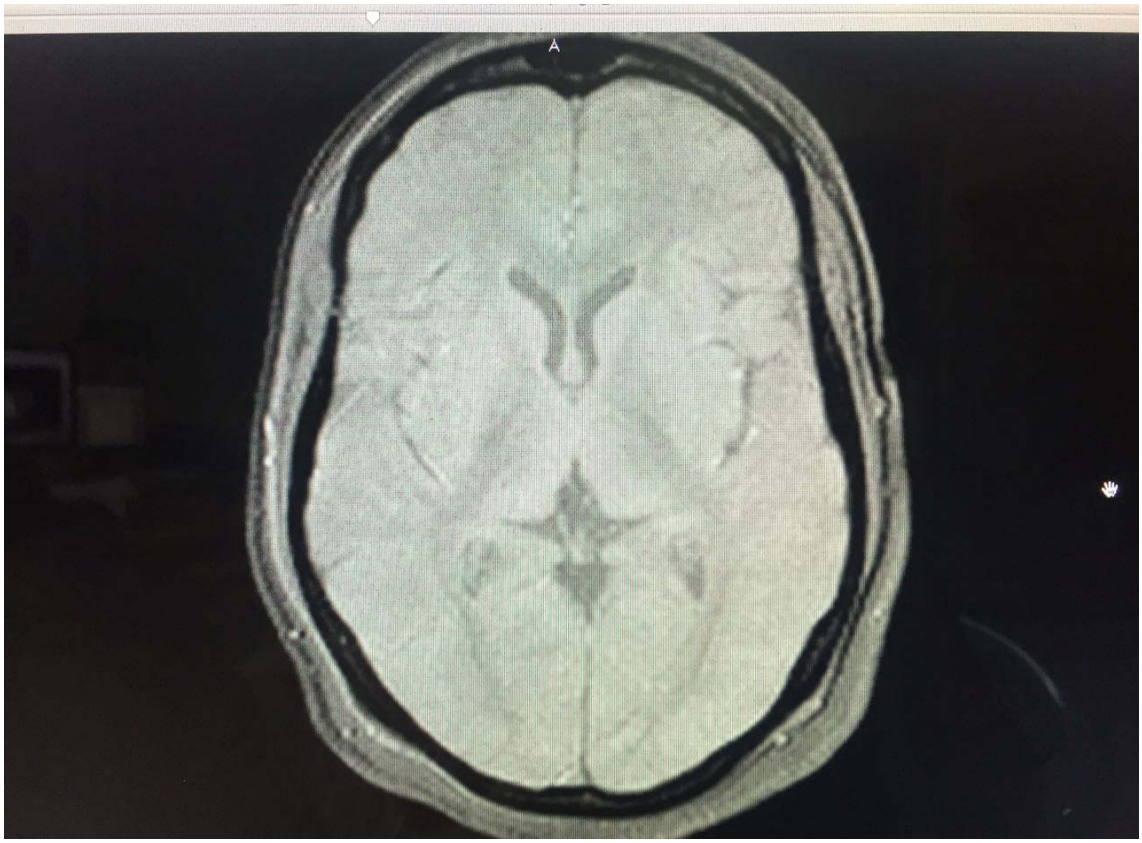
and occipital lobes. secondly at deep basal ganglia and the superior saggital sinus.

In a study done by Milliez et alno one had diffuse involvement.

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME






A

SUBARACHNOID HEMMORHAGE WITH INFARCT

SUMMARY

50 patients with eclampsia between Sep 2015- Dec 2016 at Govt.Stanley Medical College, Chennai were selected according to the inclusion and exclusion criteria already stated in the methodology were for the prospective and retrospective analytical study.

MRI Brain was done for these patient within 1 week of postpartum period .

The results were tabulated,analysed and summarized as follows.

1. Out of 50 eclampsia patients ,20%had pathological findings in the MRI of the Brain taken.
2. Majority of the patients(58%) were in the age group of (21-25 yrs)
3. Majority of the patients are multigravida(60%)
4. Majority of the patients(54%)have Normal Body Mass Index of 19.8-26.
5. Majority of the patiets are in the antepartum period (80%) at the time of presentation.
6. Majority of the patients had diagnosed hypertension(52.5%) only at the time presentation.
7. Most of the patients had imminent symptoms(90%)
8. Majority of the patients with positive MRI finding had Proteinuria of 1+
9. Most of the patients had antepartum eclampsia(80%)

10. Only in positive MRI finding group 62.5% were unconscious at the time of presentation. p value is 0.001 ,hence statistically significant.
11. In the patients with positive MRI finding ,70% had systolic blood pressure between 161-200 mmHg.p value is 0.015 which is statistically significant.
12. In the patients with positive MRI finding 40% of the patients had diastolic blood pressure in the range of 90-100mmHg. 50% had diastolic blood pressure >110mmHg.
13. 80% of the patients with positive MRI findings had MAP>126.
14. Only 10% of patients with positive MRI finding had abnormal fundus.
15. The patients with positive MRI findings had Hb% in the range of 9-11g%.
16. In the positive MRI finding group,80% of the patients have had their platelet count <3 lakh.
17. Of the patients with altered liver function test,100% had positive MRI finding, p value is 0.043,hence statistically significant.
18. Among the patients with altered renal function tests,50% had normal and abnormal MRI finding.
19. In the patients with positive MRI finding,only 40% are treated with IV Phenytoin,60% are not treated with IV Phenytoin.
20. Posterior Reversible Encephalopathy Syndrome was the most common Cerebral pathology. Most common area affected in the brain is Parieto occipital region.

CONCLUSION

1. 20% of the Eclampsia patients had pathological abnormalities detected through MRI scan of Brain.
2. Patients with altered liver function tests, normal BMI, high Systolic blood pressure, Unconscious level are prone for developing Cerebral lesions.
3. Posterior Reversible Encephalopathy Syndrome was the most common pathological abnormality detected .The next common is the Diffuse Cerebral Edema.
4. Since majority of the patients had hypertension diagnosed only at the time of presenation, the need for effective screening of hypertension and its management is emphasized.
5. Thus it is concluded that MRI brain should be included in the investigation protocol for Eclampsia if not for all, atleast for those patients with complications.

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NAME	AGE	IP NO	BMI	PARITY	GA/PP	U.ALB	HT AT	IMMI SYM	SEIZURE	PAST H/O	CONSCIOUS LEVEL	MAX BP	MAP	FUNDUS	HB%	PLATELET	LFT	RFT	MAG SULP	AC	MRI FINDIN
SARALA	30	236	25	G2P1L1		34 1+		34 H	+	NO	C	160/120	133.3	N	9	2	N	N	SD	P	N
RAMADEVI	20	3304	26.31	PRIMI		36 2+		36 NO	+	NO	C	140/100	113	N	10	3	N	N	SD	P	N
JAYANTHI	21	5614	25	PRIMI		37 1+		37 H	+	NO	D	180/130	147	N	9.7	2.7	N	N	SD	P	N
MANJULA	24	4936	25	PRIMI		37 2+		34 H	+	NO	UC	150/110	123	GR1	12	2.5	N	N	SD	P	N
HEMALATHA	20	8694	25.88	PRIMI		37 1+		37 H	+	NO	C	120/90	100	N	10.3	2.4	N	N	SD	NO	N
SUMATHI	23	3774	42	G2P1L0		34 1+		34 V	+	NO	C	160/120	133	N	9.8	2.6	N	N	SD	NO	N
SANGEETHA	24	6064	32	PRIMI		37 1+		37 NO	+	NO	D	160/90	113	N	9.6	2.8	N	N	SD	P	N
SIVARANJANI	21	2526	24.44	PRIMI		30 1+		30 NO	+	NO	C	170/120	137	N	10.5	1.25	N	N	SD	P	N
SANGEETHA	19	3113	22.48	PRIMI		33 2+		33 H+V	+	NO	C	160/120	133	N	10.2	3.2	N	N	SD	NO	N
JAYANTHI	20	3505	31.6	G2A1		35 3+		33 NO	+	NO	D	160/120	133	N	9.8	2.13	N	N	SD	NO	N
HAISHA	21	5655	24.44	PRIMI		33 T		30 NO	+	NO	C	140/100	113	N	8.2	1.2	N	N	SD	P	N
HASEENA	20	1235	25	PRIMI		36 2+		36 H	+	NO	C	160/120	133	N	9.12	1.3	N	N	SD	P	N
SUDHA	22	3088	32.47	PRIMI		37 2+		36 H+V	+	NO	C	150/90	110	N	9.8	1.5	N	N	SD	NO	N
JABAKUMARI	27	3300	31.56	G2P1L1		36 1+		36 H	+	NO	UC	170/100	123	N	8.6	1.2	N	N	SD	P	N
POOJA	19	3265	28.48	PRIMI		38 2+		37 H+V	+	NO	C	160/120	133	N	8.2	1.6	N	N	SD	NO	N
GNANASOUNDA	28	2961	32.6	G2P1L1		38 1+		38 V	+	NO	D	150/110	123	N	10	2.8	N	N	SD	P	N
KUMARI	23	4723	22.48	PRIMI		36 2+		36 H+V	+	NO	C	160/100	120	N	9.4	2.5	N	N	SD	NO	N
RAMANI	27	4234	28.57	PRIMI		35 2+		35 H	+	NO	D	160/120	133	N	9.2	1.8	N	N	SD	P	N
JEMIMA	21	3234	23.01	G2A1		31 2+		30 BOV	+	NO	C	150/100	117	N	9.8	2	N	N	SD	P	N
BALAABIRAMI	22	7312	24.45	G3P1L1A1		28 3+		28 V,H,BOV	+	NO	D	170/120	137	N	13.2	2.47	N	N	SD	NO	**F1**
EZHILARASI	19	10567	20.81	P1L1A1	PP	1+	PP	H+E	+	NO	C	160/120	133	N	7.8	1.76	N	N	SD	P	N
THAJUNISHA	24	6675	22.83	PRIMI		27 3+		28 H	+	NO	C	180/110	133	N	9.9	4	R	N	SD	P	F2+F1
SASIKALA	23	7938	29.76	G2P1L1		38 1+		38 H	+	NO	C	160/110	127	N	12.6	1.92	N	N	SD	P	N
VAIJAYANTHI	21	7391	28.31	G2A1		32 2+		32 V	+	NO	D	160/120	133	N	9.2	2.1	N	N	SD	P	N
HEMAVATHY	23	3836	29.7	P1L1	PP	1+	PP	H	+	NO	C	170/110	130	N	11.6	2.82	N	N	SD	P	N
VANITHA	34	7023	23.24	P2L2	PP	2+	PP	H	+	NO	D	160/120	133	N	8.6	1.78	N	N	SD	NO	**F1**
SOUNDARYA	18	9701	20.41	P1L1	PP	1+	PP	H	+	NO	D	170/100	123	N	12.1	2.54	N	N	SD	NO	**F4+F5**
RESHMA	27	11078	23.19	G3P1L1A1		34 2+		32 H	+	NO	UC	160/120	133	N	10.2	2.4	N	N	SD	P	F3
SOUNDARYA	21	8297	22.84	P1L1	PP	2+	PP	V	+	NO	UC	140/100	113	N	9.2	2.5	N	N	SD	NO	**F1**
HARITHA	19	9814	25.43	PRIMI		36 1+		36 V	+	NO	C	160/120	133	N	9.7	2.4	N	N	SD	P	N
SHANVAS	18	3932	19.81	P1L1	PP	1+	PP	H	+	NO	UC	190/130	150	N	9.2	1.5	N	N	SD	P	**F1**
PONNI	22	4113	24.56	P1L1	PP	3+	PP	H	+	NO	C	160/120	133	N	9.5	2.15	N	N	SD	NO	N
SARANYA	26	4556	25.3	P2L3	PP	2+	PP	H	+	NO	D	180/110	133	N	10.1	2.5	N	N	SD	P	N
VIDHYA	20	5221	26.04	PRIMI		36 1+		34 H+V	+	NO	C	150/90	110	N	12.3	2.6	N	N	SD	NO	N
BOOMA	23	4576	26.84	G2P1L1		28 2+		28 H	+	NO	UC	180/100	127	N	10.4	3.4	N	N	SD	P	**F1**
KAUSALYA	24	6767	23.37	G2A1		35 1+		32 H	+	NO	C	160/120	133	N	11	3.2	N	N	SD	NO	N
PRIYA	22	3431	25.79	G2P1L1		36 2+		37 H+V	+	NO	C	150/100	117	N	10.6	2.3	N	N	SD	NO	N
JEEVITHA	28	5621	20	G3P1L1A1		34 2+		36 V	+	NO	C	150/110	123	N	9.4	2.1	N	N	SD	NO	N
DIANA	23	3432	20.31	G2A1		30 3+		30 BOV	+	NO	D	160/120	133	N	10.1	1.6	N	N	SD	P	N
VINODHA	21	8767	24.84	PRIMI		37 1+		36 H	+	NO	C	150/120	130	N	9.2	1.5	N	N	SD	P	N
GEETHA	22	4443	23.62	G2A1		36 1+		32 H	+	NO	UC	180/100	127	GR1	9.4	1.1	N	N	SD	NO	**F2**
ANITHA	23	1112	21.93	P2L2	PP	2+	PP	H+V	+	NO	C	160/120	133	N	9.2	2.3	N	N	SD	P	N
LAVANYA	18	2334	23.01	PRIMI		32 1+		30 H	+	NO	UC	190/100	130	N	9.9	2.5	N	N	SD	P	N
PARVEEN	24	2465	22.43	G3P1L1A1		34 2+		32 H	+	NO	C	140/100	113	N	10.4	3.2	N	N	SD	NO	N
MONISHA	25	2112	20.58	P2L1	PP	1+	PP	V	+	NO	C	200/120	147	N	11.1	2.3	N	N	SD	P	N
DEIVANAI	24	2133	21.09	G2P1L1		24 2+		24 H	+	NO	C	160/120	133	N	10.2	1.8	N	N	SD	P	N
RADHA	26	2213	21.63	PRIMI		32 1+		30 H+V	+	NO	D	180/120	140	N	12	1.3	N	N	SD	NO	**F2**
VICTORIA	25	12086	33.29	G3P2L2		33 3+		33 V	+	NO	D	170/100	123	N	10.4	1.7	N	N	SD	NO	N
KAMATCHI	21	12176	21.34	G2A1		38 1+		35 H	+	NO	C	160/110	127	N	11	2.3	N	N	SD	P	N
DIVYA	22	12090	23.74	PRIMI		38 2+		33 H	+	NO	C	180/110	133	N	10.2	2.24	N	N	SD	NO	N

சுய ஒப்புதல் படிவம்

வலிப்பு வந்த கர்ப்பிணி பெண்களுக்கு மூளைக்கு செயல்படும் எம்.ஆர்.ஐ. ஸ்கேன் (காந்த ஒத்ததிர்வு தோற்றுருவாக்கல்) பயன் பற்றி கண்டறிதல்

ஆய்வாளர் : மரு. அ. சி. கிரிதா ரஞ்சனி,
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பெயர் : வயது : உள்ளிருப்பு எண் :

இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது என்னுடைய சந்தேகங்களை தீர்க்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

இந்த ஆய்விற்காக எனக்கு எடுக்கப்பட்ட எம்.ஆர்.ஐ. மூளைப்பகுதியின் ஸ்கேனின் பரிசோதணை அறிக்கையை மருத்துவ ஆய்விற்காக பயன்படுத்திக்கொள்ள சம்மதிக்கிறேன்

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும் எந்த கட்டத்திலும் எந்த சட்டசிக்கலும் இன்றி இந்த ஆய்விலிருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

நான் ஆய்விலிருந்து விலகிக்கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவையில்லை எனவும் அறிந்து கொண்டேன் என்னை பற்றிய தகவல்கள் ரகசியமாக பாதுகாக்ப்படும் என்பதையும் அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் ஆய்வாளர் அவர் விருப்பத்திற்கேற்ப பயன்படுத்திக் கொள்ளவும் அதனை பிரசுரிக்கவும் முழுமனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன் எனக்கு கொடுக்கப்பட்டுள்ள அறிவுரைகளின்படி நடந்து கொள்வதுடன் ஆய்வாளருக்கு உண்மையுடன் இருப்பேன், என்றும் உறுதி அளிக்கிறேன்.

உடல்நலம் பாதிக்கப்பட்டாலோ வழக்கத்திற்கு மாறான ஏதேனும் நோய்குறி தென்பட்டாலோ அதனை தெரிவிப்பேன் என்றும் உறுதி கூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்விதமான பரிசோதனைகளையும் சிகிச்சைகளையும் மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வுக்கு தேவைப்பட்டால் முதலமைச்சரின் மருத்துவ காப்பீட்டுத்திட்டத்தில் இருந்து நிதி உதவியை பயன் படுத்திக் கொள்ள சம்மதிக்கிறேன்.

இப்படிக்கு,

ஆய்வாளரின் கையொப்பம் நோயாளியின் கையொப்பம்

CONSENT FORM

I agree to participate in the study entitled '**ANALYTICAL STUDY OF MRI BRAIN IN ANTEPARTUM AND POSTPARTIM ECLAMPSIA PATIENTS**"

I agree to give my MRI Brain report for this study

I confirm that I have been told about this study in my mother tongue and have had the opportunity to clarify my doubts.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reasons and without affecting my benifits.

I agree not to restrict the use of any data or results that arise from this study.

Name of the participant :

Sign / Thumb print:

Sign of Investigator :

PROFORMA

DATE:

NAME: AGE: LMP:

IP NO: EDD:

D.O.A: D.O.D OBSTETRIC

CODE:

ADDRESS & CONTACT NO:

PRESENTING COMPLAINTS:

MENSTRUAL HISTORY : MD SINCE:

OBSTETRIC HISTORY:

PAST HISTORY:

GENERAL EXAMINATION:

HT: WT: BMI: TEMP: PR: BP:

PALLOR: PEDAL EDEMA:

CVS: RS:

P/A:

CNS:

Pupils:

GCS: Reflexes:

Deep tendon reflexes:

Plantar Reflex:

INVESTIGATIONS:

COMPLETE HEMOGRAM:

Hb

Packed cell volume:

Tc: Dc: Platelets:

RBC:

WBC:

Random blood sugar:

RENAL FUNCTION TEST:

BLOOD UREA:

SERUM CREATININE:

LIVER FUNCTION TEST:

TOTAL BILIRUBIN: DIRECT BILIRUBIN: INDIRECT BILIRUBIN:

SGOT: SGPT: SAP: TOTAL PROTEIN:

SERUM ALBUMIN:

COAGULATION PROFILE:

BLEEDING TIME:

CLOTTING TIME:

SERUM FIBRINOGEN:

PT: INR: aPTT:

SERUM ELECTROLYTES:

SODIUM:

POTASSIUM:

MRI BRAIN:

TREATMENT:

STAY IN HOSPITAL:

OUTCOME:

RESULT:

ABBREVIATIONS

BP	-	Blood Pressure
MRI	-	Magnetic Resonance and Imaging
ALT	-	Alanine Transaminase
AST	-	Aspartate Transaminase
PGI2	-	Prostaglandins
ROS	-	Reactive Oxygen Species
VCAM	-	Vascular Cell Adhesion Molecule
HLA	-	Human Leucocyte Antigen
DIC	-	Disseminated Intravascular Coagulation
CT	-	Computed Tomography
SPECT	-	Single Photon Emission Computed Tomography
DWI	-	Diffusion Weighted Images
ADC	-	Apparent Diffusion Coefficient
AT II	-	Angiotensin II
MAP	-	Mean Arterial Pressure
BMI	-	Body Mass Index
LFT	-	Liver Function Test
RFT	-	Renal Function Test

KEY TO MASTER CHART

H	HEADACHE
V	VOMITING
E	EPIGASTRIC PAIN
BOV	BLURRING OF VISION
D	DROWSY
UC	UNCONSCIOUS
C	CONSCIOUS
N	NORMAL
SD	STANDARD DOSE REGIMEN
GA	GESTATIONAL AGE
PP	POSTPARTUM
HT AT	HYPERTENSION AT
U.ALB	URINE ALBUMIN

MAG.SULP -MAGNESIUM SULPHATE

AC-ANTICONVULSANT

MAP-MEAN ARTERIAL PRESSURE

P-PHENYTOIN

IMM- IMMINENT SYMPTOM

F1- PRES (Posterior Reversible Encephalopathy Syndrome)

F2-DIFFUSE CEREBRAL EDEMA+PRES

F3-RIGHT TEMPORAL INFARCT

F4-ACUTE INFARCT + SUBARACHNOID HEMMORHAGE